# ASPHYXIATION, SUFFOCATION, AND NECK PRESSURE DEATHS

EDITED BY

Burkhard Madea

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# Preface

Asphyxia-related deaths are common in daily forensic casework: many suicides and homicides as well as accidents can be attributed to asphyxia. However, it is well known that there are a significant number of unrecorded cases of homicide by asphyxia because the macromorphological findings may be weak or even absent or may be misinterpreted.

One of the most famous criminal cases in Germany in the middle of the last century was a so-called case Hetzel. Hetzel was accused of having killed a young woman by neck compression for sexual reasons. Unfortunately, the initial autopsy was carried out by clinical pathologists not forensic pathologists. The clinical pathologists were unfamiliar with morphological findings in cases of strangulation. Based on the autopsy findings and later police investigations, the famous specialist in forensic medicine at the University of Münster, Albert Ponsold, together with his deputy Walter Krauland, submitted a written report on the cause of death and they concluded death was due to strangulation.

I would have no problem in signing this report today, 60 years after it was originally written. Both experts made the best of deficient autopsy descriptions.

At court, Ponsold specified the kind of strangulation as ligature strangulation and even identified, based on patterned abrasions on the skin of the neck, the kind of rope: a 'Kälberstrick' (veal rope). The veal rope gave the trial the name ('Kälberstrick-Prozess', 'veal-rope trial').

The accused was sentenced to life-long imprisonment for first degree murder.

Ten years later, there was a new retrial. Several other experts had prepared written reports for the defense and one claimed that the dried abrasion on the neck was not due to ligature strangulation but to lying of the skin on a wooden stick in the road ditch where the body was found. The abrasion was, in his opinion, a postmortem abrasion and cause of death was due to heart failure during sexual intercourse (in this 23-year-old female!!).

In the retrial, the original expert Prof. Ponsold was not heard from again, but only those who opposed his opinion on the cause of death. After the retrial, the imprisoned Hetzel was found innocent and released from prison immediately. This acquittal following the retrial was, of course, a mistake based on expert opinions which had the primary aim of destroying the position of Prof. Albert Ponsold as "pope" of Germany forensic medicine. From this case much can be learned:

- The first point is that forensic autopsies should be carried out by qualified and board-certified forensic pathologists and not by clinical pathologists.
- Of course, a strangulation mark can also be produced postmortem; it is in itself not a vital sign. The vitality is proofed by other vital reactions. However, if a postmortem development is claimed by lying on a wooden branch of a tree, similar abrasions should also be visible on other body parts, especially prominent parts of the face. This was obviously not the case.
- Of course, all findings have to be seen together to come to a definite conclusion. When the strangulation mark was produced postmortem due to lying on the branch of a tree and petechial hemorrhages also developed postmortem due to a slight head down position, the correct diagnosis will be missed.
- Heart failure during sexual intercourse in a young swinging woman is a strange cause of death with no proof by morphological findings.
- The case shows that the diagnosis of fatal asphyxiation may be difficult, even today.

The main contributions to the understanding of fatal asphyxia and asphyxia-related deaths had been made in the second half of the 19th century. Many very important experiments on human bodies had been carried out already in the 19th century, f.i. by Eduard von Hofmann and his school (not only on hanging, but also on drowning (Arnold Paltauf) and carbon monoxide asphyxiation during fire (so called Wiener Ringtheater Brand/Ring theatre fire, Eduard Zillner).

Self-experiments on the velocity of loss of consciousness in hanging and ligature strangulation had been carried out in the first half of the 20th century. Later biochemical vital reactions have been studied extensively.

Further micro-morphological reaction patterns, for example, pulmonary giant cells or the pulmonary hemorrhagic-dysoric syndrome have also been described which are, however, discussed controversially in the literature.

Based on a systematic analysis of video recordings of human hangings and the creation of the working group on human asphyxia, new data on the pathophysiology of hanging and the agonal sequence in hanging were obtained which also led to a new proposal of classification of asphyxia.

There are unfortunately different classifications of asphyxia deaths and there is a need for standardization.

Already in the 19th century asphyxia-related deaths were part of famous textbooks which, even today, are worthy of reading due to the unique case reports and illustrations.

Several monographs on asphyxiation were published, f.i. by Tardieu, Brouardel, Minovici, Brinkmann and Püschel, Sauvageau and Geberth. The monograph of Sauvageau and Geberth on autoerotic deaths presents a scientific modern view of autoerotic deaths and has, of course, many overlaps with this book on asphyxia deaths.

The aim of this book is to give an up-to-date review of the knowledge on asphyxia-related deaths. Open questions shall be addressed as well as controversial findings. The contributions tried to include the most recent developments of research on asphyxia-related deaths into this book. And this is indeed what this book is all about: presenting a scientific modern view of asphyxia-related deaths.

Authors from ten countries (Canada, Finland, Germany, Italy, Japan, Romania, Sweden, Switzerland, the U.K., and the United States) and three continents (Europe, North America, Asia) contributed to the book.

Of course, in the preparation of a book with so many authors there are always delays. Therefore, I am glad that it has nevertheless finally reached fruition. I am very much in the debt of my contributors for their input and, of course, to the publisher for making the publication of this book possible.

**Burkhard Madea**

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# 1

# History of Asphyxia-related Deaths

*Burkhard Madea*

## Historical background

### Development of forensic medicine

#### Introduction

According to Sydney Smith (1951), forensic medicine may be defined briefly as consisting essentially of that body of medical and paramedical scientific knowledge which may be used for the purposes of administration of the law. Alfred Swaine Taylor (1844) has defined medical jurisprudence as 'that science, which teaches the application of every branch of medical knowledge to the purpose of the law'. According to a German definition by Schmidtmann (1907), the last editor of the famous *Handbook of Forensic Medicine* of Johann Ludwig Casper, forensic medicine is a cross-sectional discipline of medicine and natural sciences dealing with all medical evidence that is relevant for law. It deals with medical evidence not only in practice but also in research and, furthermore, all legal essentials in healthcare, especially for doctors, are part of teaching, training and research.

There is, of course, no specific date on which forensic medicine emerged as a recognizable separate scientific discipline. Several steps in the development of forensic medicine can be distinguished (Table 1.1): first, the use of medical knowledge for legal and public purposes; second, the compulsory medical testimony for the guidance of judges in special cases; and third, the professionalization as a named discipline.

Forensic medicine is a speciality: how we experience it at the beginning of the 21st century is the result of developments since the 19th century. Of course, forensic medicine has much older roots. Often the famous criminal code of Emperor Charles V, the *Constitutio Criminalis Carolina,* promulgated in 1532, has been called the first important landmark in the history of legal medicine (Figure 1.1). Brittain (1965) wrote:

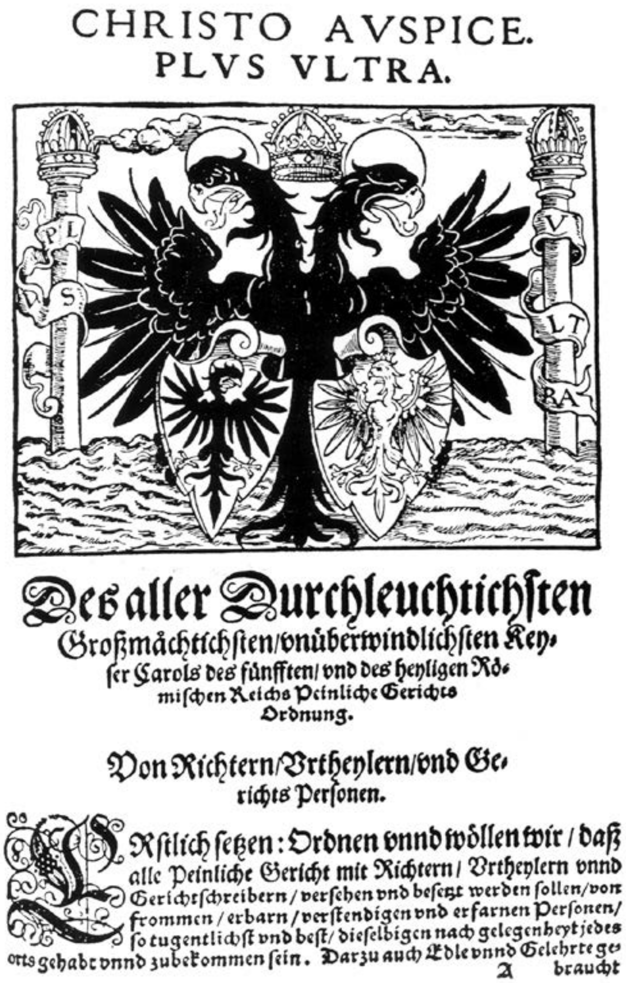
> 'It has commonly been considered as the true start of legal medicine, and hence Germany has been hailed as the country which gave birth to the discipline. It has been said that it caused medical men to be called in for legal matters for the first time. This is not strictly true. They had been called on before as earlier enactments show. Without in any way minimising the advance the Carolina represented, it would be wrong to consider it as a phenomenon which occurred without logical antecedents, and by implication that legal medicine arose by a kind of spontaneous generation.'

According to Bertrand Ludes (2008, 2017), modern forensic medicine was born in France during the French Revolution with the closure of old universities and the creation of three new faculties of medicine in Paris, Strasbourg and Montpellier. Medical studies were reorganized in 1794 and professorships of forensic medicine were established in the new faculties. In 1789 François-Emmanuel Fodéré (1764–1835) published his legislation enlightened by physical sciences, or treatises of forensic medicine in public health, which represented the first French publication with forensic medicine in its title.

Other famous forensic scientists were Alphonse Devergie (1798–1879), author of a monumental treatise in 1853, Paul C. H. Brouardel (1837–1902), and Ambroise Auguste Tardieu

**Table 1.1 Development of forensic medicine**

| Step | Description |
|---|---|
| 1 | Medical knowledge is used for legal or public purposes. Dependent on point of achievement in both law and medicine:<br>• knowledge of medical plants, botany<br>• knowledge of injuries<br>• educational standards in medicine<br>• standards of competency<br>• legislation concerning disposal of the dead<br>• legislation concerning injuries<br>• compensation for injuries and deaths. |
| 2 | Expert medical testimony must be obtained for the guidance of judges in cases of murder, wounding, poisoning, hanging, drowning, infanticide, abortion, malpractice. |
| 3 | Further professionalization:<br>• medicolegal examination<br>• giving evidence at court/medical expertise required at court<br>• publication of monographs<br>• teaching<br>• systematic research (decrease of the domain of magic and sorcery)<br>• knowledge gained by own practice replaces textbook knowledge (J. L. Casper)<br>• foundation of professorships<br>• foundation of own institutes<br>• foundation of societies. |



CHRISTO AVSPICE.
PLVS VLTRA.

Des aller Durchleuchtichſten
Großmächtichſten/vnübertwindlichſten Keyſer
Carols des fünfften/ vnd des heyligen Römiſchen
Reichs Peinliche Gerichts
Ordnung.

Von Richtern/Vrtheylern/vnd Gerichts Perſonen.

ERſtlich ſetzen: Ordnen vnnd wöllen wir / daß alle Peinliche Gericht mit Richtern / Vrtheylern vnnd Gerichtſchreibern / verſehen vnd beſetzt werden ſollen/ von frommen / erbarn / verſtendigen vnd erfarnen Perſonen/ ſo tugentlichſt vnd beſt/ dieſelbigen nach gelegenheyt jedes orts gehabt vnnd zubekommen ſein. Darzu auch Edle vnnd Gelehrte ge-
A braucht

**Figure 1.1** *Constitutio Criminalis Carolina* (criminal code of Emperor Charles V).

**Figure 1.2** Johann Ludwig Casper (1796–1864), founder of modern forensic medicine in Prussia.

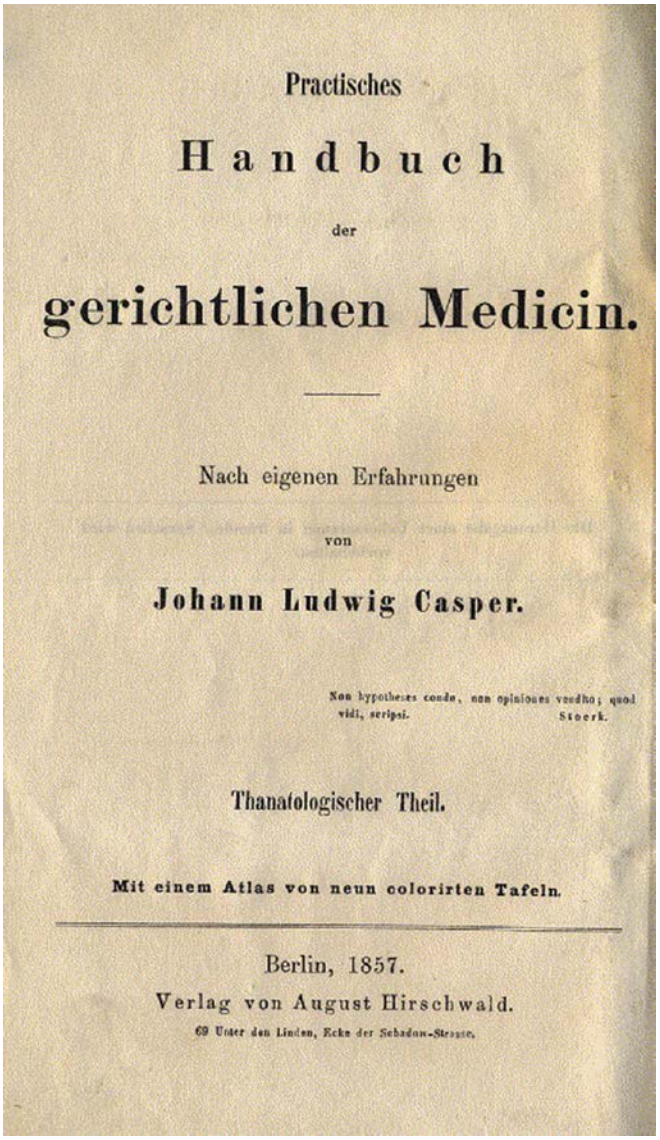
(1818–1879), who was a pupil of Orfila and, like his master, a courtroom star. They no longer produced 'treatises', but special monographs on particular issues such as hanging, abortion, poisoning and wounds. Tardieu wrote the first book on sexual abuse of children and on battered children. Subpleural haemorrhages are named after him. Brouardel held the Chair of Forensic Medicine in Paris between 1879 and 1896 and also became Dean of the Faculty of Medicine.

One of the most remarkable experts in forensic medicine in the 19th century was Johann Ludwig Casper (1796–1864), the founder of modern forensic medicine in Prussia (Figure 1.2).

In 1852, Casper founded the quarterly *Journal of Forensic and Public Medicine* and, in 1857, the first edition of his *Practisches Handbuch der Gerichtlichen Medicin* (Practical Handbook of Forensic Medicine) was published (Figure 1.3). This handbook, which was also translated into English, was revolutionary since its content was based on the author's own observations. His motto was: *'Non hypotheses condo, non optiones vendito, quod vidi scripsi'*.

In 1804, in Vienna, an institute of forensic medicine was founded as an institute of forensic pharmacology and medical police. However, from 1844 to 1875, forensic autopsies were performed by pathologists, mainly by the famous pathologist Carl von Rokitansky (1804–1878). In 1875, Eduard von Hofmann (1837–1897) (Figure 1.4a), who was the first Professor of Forensic Medicine at the University of Innsbruck since 1869, moved to Vienna. In 1878, he published his famous *Lehrbuch der Gerichtlichen Medicin* (Textbook of Forensic Medicine), which was translated into four languages (French, Russian, Italian and Spanish) (Figure 1.4b). Table 1.2 lists famous textbooks in forensic medicine in the 19th and early 20th centuries. He also published a famous *Atlas der Gerichtlichen Medizin* (Atlas of Forensic Medicine) (Figure 1.4c) with remarkable drawings (Figure 1.5a,b).

Forensic medicine in the modern sense developed during the second half of the 19th century. Autopsies, the systematic evaluation of autopsy results and animal experiments were the main research methods and they



Practisches
Handbuch
der
gerichtlichen Medicin.

Nach eigenen Erfahrungen
von
Johann Ludwig Casper.

Non hypotheses condo, non opiniones vendito; quod vidi, scripsi.
Stoerck.

Thanatologischer Theil.

Mit einem Atlas von neun colorirten Tafeln.

Berlin, 1857.
Verlag von August Hirschwald.
69 Unter den Linden, Ecke der Schadow-Strasse.

**Figure 1.3** Johann Ludwig Casper's *Practisches Handbuch der Gerichtlichen Medicin* 1st ed., published in 1857.

remained the principal form of medical research in the 19th and early 20th centuries. Many diseases have been discovered or critically clarified through autopsy or observations and experiments carried out on the deceased. Furthermore, experiments on human bodies were essential for the understanding of asphyxia deaths.

In the second part of the 19th century, Forensic Medicine developed as a scientific discipline in the modern sense, based on the progress of natural sciences.

## Asphyxia-related deaths: Early history

Long before oxygen and circulation were detected, people knew that strangulation would cause death. Illustrations of suicidal hanging can be found on a capital in the famous church of Saint Mary Magdalene in Vézelay, France (suicide of Judas Iscariot), and in a famous drawing by Leonardo da Vinci showing the hanging of Bernardo Bandini (1421–1479) in the Bargello in Florence (Figures 1.6 and 1.7). Bandini participated in the assassination of Giuliano de Medici in Florence Cathedral.

Up until the 19th century, a common theory of different types of pathogenesis in different types of asphyxia was missing. The lack of understanding of asphyxia was the reason why causes of death which had nothing to do with asphyxia were attributed to asphyxia while others which were asphyxial were excluded. Indeed, although a clear differentiation and terminology of asphyxial deaths was achieved in the first part of the 20th century, new proposals are still being made at the beginning of the 21st century.

The historian of medicine Esther Fischer-Homberger gave a review of the history of various manners of death, among them asphyxiation, in her book *Medizin vor Gericht. Zur Sozialgeschichte der Gerichtsmedizin* (Medicine at Court. On the Social History of Forensic Medicine) (1988).

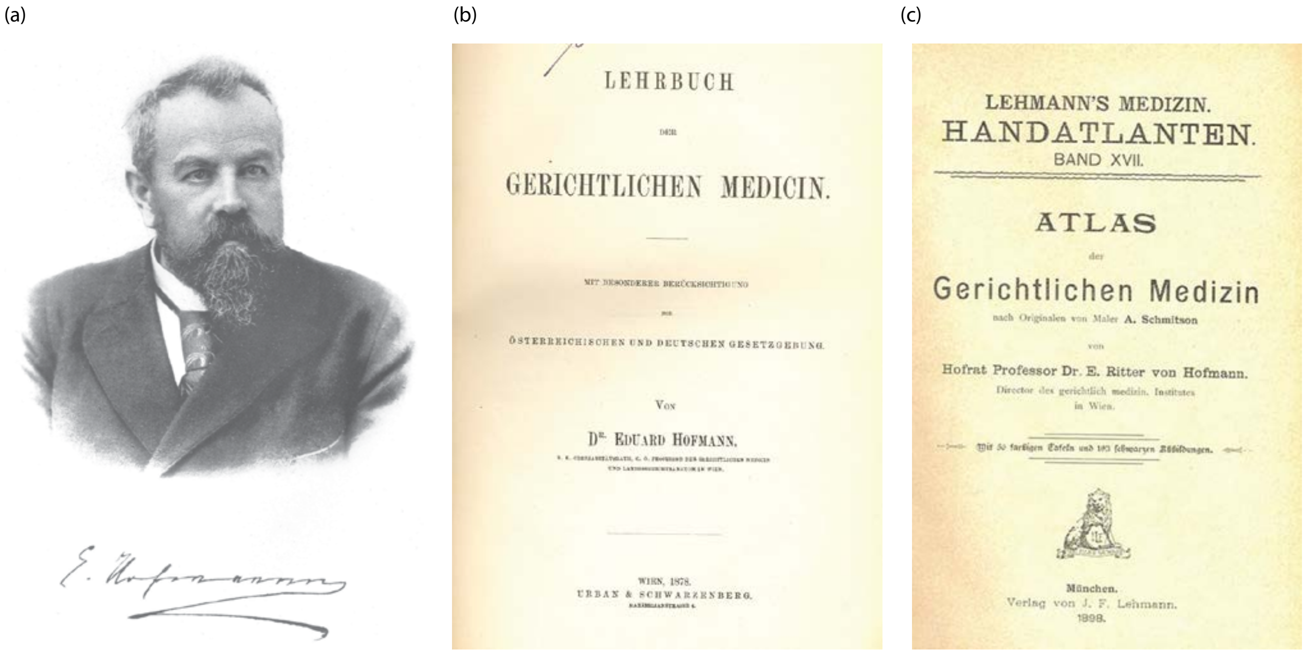
Long before William Harvey (1578–1657) discovered blood circulation and Antoine-Laurent Lavoisier (1743–1794) oxygen, it was known that occlusion of respiratory orifices or strangulation would cause death. With the discovery of oxygen by Lavoisier, the foundation stone of modern respiratory physiology was laid (composition of air, respiration, oxidation, metabolism) (Table 1.3). The medicolegal experts of the 16th to 18th centuries did not recognise the entity 'asphyxiation' although drowning, ligature strangulation, hanging, death due to asphyxiation or in life-threatening atmospheres were known to them.

Before Lavoisier, oxygen deficiency was not recognized as a cause of death, not even as 'air-deficiency' because the composition of air was not known at that time. Air was not yet recognized as 'air vital' but was considered as 'pneuma' that was necessary for the heart to produce spirits of life (spiritus vitalis) which were distributed by the arteries. However, it was already known that, in some places, survival may be impossible – for instance, in the vicinity of glowing charcoals or in the so-called 'Caves of Choron'. Erasistratos (born ca. 304 BC) explained that the air was too thin for the body. Galen (129–199/200/216) refused this interpretation. For him, the quality of air was just unpleasant.

Ambroise Paré (1510–1590) reported on the successful resuscitation of two servants who were found unconsciousness in a charcoal-poisoned atmosphere. Death due to charcoal was thought to be due to a 'constipation of brain and nerves', as in a stroke.

As early as 1842, the chemist Felix Leblanc described carbon monoxide (CO) poisoning. Under Eduard von Hofmann's direction, the victims of the Ring Theatre fire (8 December 1881) were forensically examined. Together with his pupil Eduard Zillner, he could demonstrate that the presence of carbon monoxide haemoglobin in the blood of fire victims proved the vital inhalation of fumes.

Ambroise Paré addressed already vital reactions in hanging. As a vital sign, a strangulation mark corresponding



LEHRBUCH
DER
GERICHTLICHEN MEDICIN.
MIT BESONDERER BERÜCKSICHTIGUNG
DER
ÖSTERREICHISCHEN UND DEUTSCHEN GESETZGEBUNG.
VON
DR. EDUARD HOFMANN,
WIEN, 1878.
URBAN & SCHWARZENBERG.

LEHMANN'S MEDIZIN.
HANDATLANTEN.
BAND XVII.
ATLAS
der
Gerichtlichen Medizin
nach Originalen von Maler A. Schmitson
von
Hofrat Professor Dr. E. Ritter von Hofmann.
Director des gerichtlich medizin. Institutes
in Wien.
Mit 56 farbigen Tafeln und 193 schwarzen Abbildungen.
München.
Verlag von J. F. Lehmann.
1898.

**Figure 1.4** (a) Eduard von Hofmann (1837–1897), Professor of Forensic Medicine in Vienna from 1875 to 1887. His time in Vienna is called the golden age of forensic medicine. He published not only a famous textbook and an atlas of forensic medicine, but also numerous articles throughout the whole discipline. He had many important pupils who performed outstanding experimental research. (b) Title page of Eduard von Hofmann's 1878 *Textbook of Forensic Medicine.* (c) Title page of Eduard von Hofmann's 1898 *Atlas of Forensic Medicine*.

**Table 1.2 Famous textbooks on forensic medicine in the 19th and early 20th centuries**

| Author | Publication |
|---|---|
| Caspar, Johann Ludwig | *Practisches Handbuch der Gerichtlichen Medicin*, 1st ed. 1857, 9th ed. edited by R. Schmidtmann 1905 |
| Von Hofmann, Eduard | *Lehrbuch der Gerichtlichen Medicin*, 1st ed. 1878, 11th ed. edited by Albin Haberda 1923 |
| Von Hofmann, Eduard | *Atlas der Gerichtlichen Medizin*, 1898 |
| Ponsold, Albert | *Lehrbuch der Gerichtlichen Medizin*, 1st ed. 1950, 3rd ed. 1967 |
| Prokop, Otto | *Forensische Medizin*, 1st ed. 1960, 3rd ed. 1975 |
| Mueller, Berthold | *Gerichtliche Medizin*, 1st ed. 1953, 2nd ed. 1975 |
| Strassmann, Fritz | *Lehrbuch der Gerichtlichen Medizin*, 1st ed. 1895, 2nd ed. 1931 |
| Brouardel, Paul | *L'Infanticide*. Paris, J.-B. Baillière et Fils, 1897 |
| Von Hofmann, Eduard | *Nouveaux éléments de médicine légale*, traduction par le Dr. Emmanuel Lévy; introduction et commentaires par P. Brouardel. Paris, J.-B. Baillière et Fils, 1881 |
| Von Hofmann, Eduard | *Atlas-manuel de médicine légale*, édition française par Ch. Vilbert; introduction par P. Brouardel. Paris, J.-B. Baillière et Fils, 1899 |
| Lacassagne, Alexandre | *Précis de médicine judiciare*, ouvrage accompagné de 4 figures dans le texte et de 4 planches en couleur dessinées par le Dr. E. Charvot. Paris, G. Masson, 1878 |
| Lacassagne, Alexandre | *Le Vade-mecum du médicin-expert : guide médical ou aide-mémoire de l'expert, du juge d'instruction, des officiers de police judicaire, de l'avocat*. Lyon, A. Storck, 1892 |
| Brouardel, Paul | *La pendaison, la strangulation, la suffocation, la submersion*. Paris, J.-B. Baillière et Fils éditeurs, 1897 |
| Orfila, Mathieu-Joseph | *Leçons de médicine légale*. Paris, Bechet Jeune Editeur, 1828 |
| Orfila, Mathieu-Joseph, Lesueur, Octave | *Traité des exhumations juridiques et considérations sur les changements physiques que les cadavres éprouvent en se pourrissant dans la terre, dans l'eau*. Paris, Béchet Jeune, Libraire de la Faculté de Médicine, 1831 |
| Simonin, Camille | *Médicine légale judiciaire*, 3rd ed. Collection Les Précis Pratiques, Paris, Maloine, 1955 |
| Taylor, Alfred S. | *A Manual of Medical Jurisprudence*. London, John Churchill, 1844 |
| Tardieu, Ambroise, avec la collaboration de Z. Roussin | *Étude médico-légale et clinique sur l'empoisonnement*. Paris, J.-B. Baillière et Fils, 1867 |
| Tardieu, Ambroise | *Étude médico-légale sur l'infanticide*. Paris, J.-B. Baillière et Fils, 1868 |
| Tardieu, Ambroise | *Étude médico-légale sur la pendaison, la strangulation et la suffocation*, 2nd ed. Paris, J.-B. Baillière et Fils, 1879 |
| Minovici, Nicolae | *Étude sur la pendaison*. Bibliotheque de Criminologie, Paris, Maloine, 1905 |
| Reuter, Fritz | *Lehrbuch der Gerichtlichen Medizin*. Berlin, Urban & Schwarzenberg, 1933 |

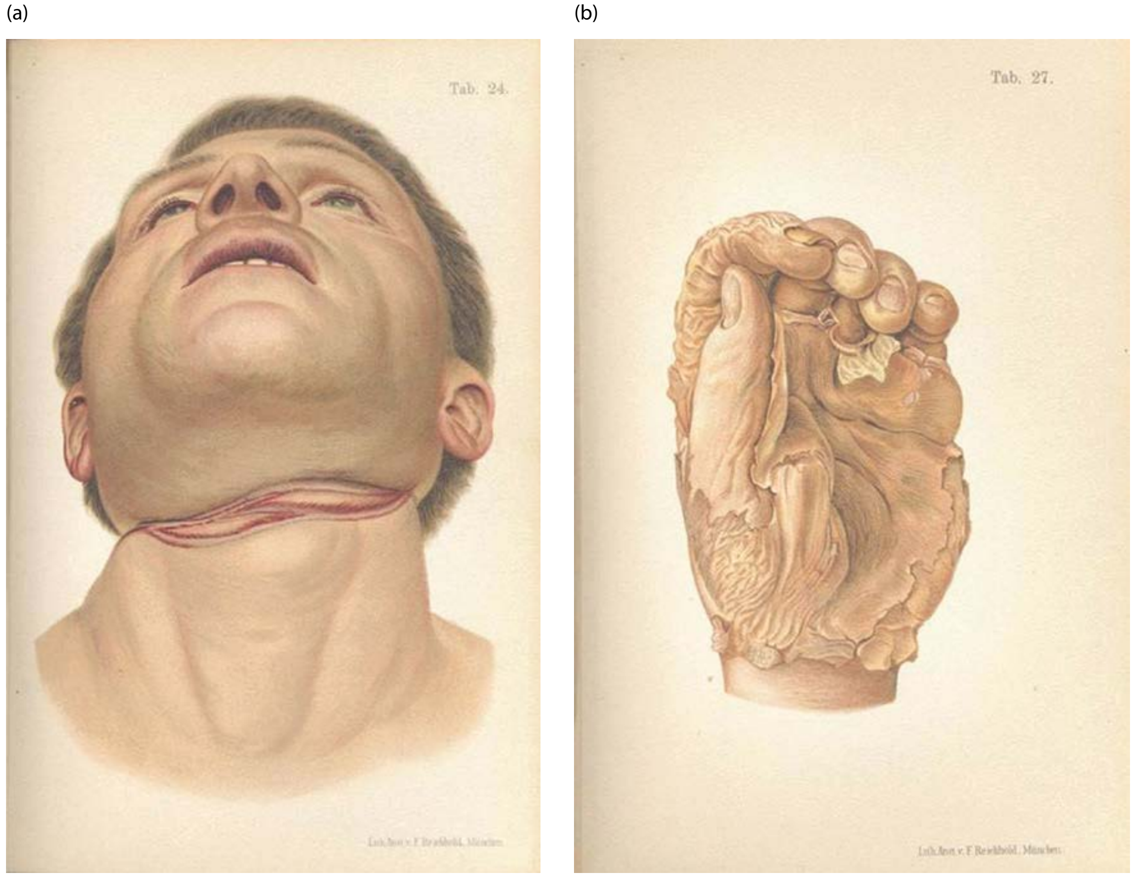

(a)
(b)
Tab. 24.
Tab. 27.
Lith.Anst.v.F.Reichhold, München
Lith.Anst.v.F.Reichhold, München

**Figure 1.5** (a), (b) Illustrations from Eduard von Hofmann's *Atlas of Forensic Medicine*.

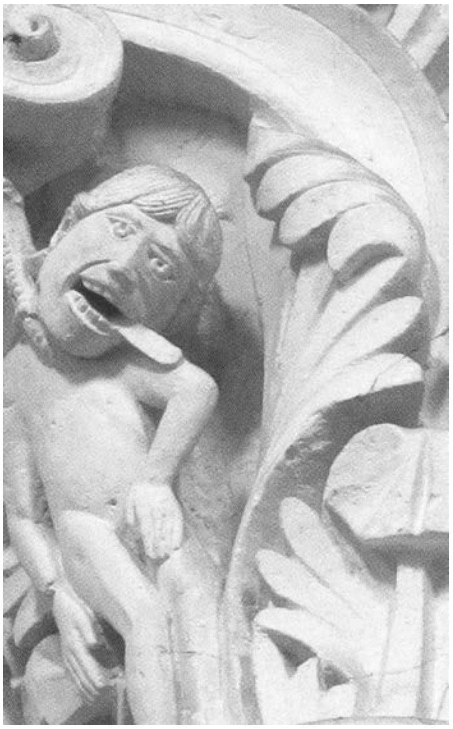


**Figure 1.6** Suicide of Judas Iscariot: capital in the church of Saint Mary Magdalene in Vézelay, France.

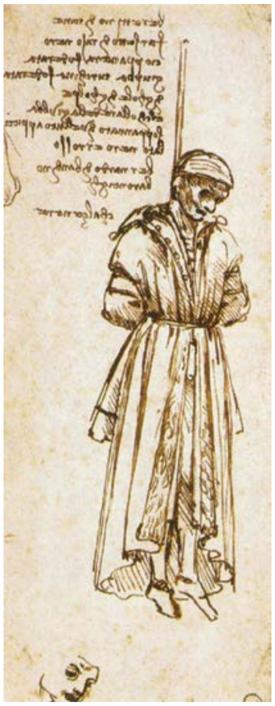


**Figure 1.7** Leonardo da Vinci drawing of the hanging of Bernardo Bandini (1421–1479) after the assassination of Giuliano de Medici, Bayonne, Musée Bonnat.

**Table 1.3 Some steps in understanding the physiology of asphyxiation**

| Forensic scientist | Area of study |
|---|---|
| William Harvey (1578–1657) | Discovery of circulation<br>*De motu cordis* (1628) |
| Antoine-Laurent Lavoisier (1743–1794) | Discovery of oxygen, combustion, respiratory physiology |
| Eduard von Hofmann (1837–1897) and pupils | Importance of cessation of cerebral circulation in hanging |
| Ecker (1870) | Airway obstruction in strangulation |
| Heinrich Ewald Hering (1866–1948) | Carotis sinus reflex<br>*Die Karotissinusreflexe auf Herz und Gefäße von normal-physiologischen, pathologisch-physiologischen und klinischen Standpunkt.* Dresden, Steinkopff, 1927 |
| Nicolae Minovici (1868–1941) | Studies on loss of consciousness in hanging |
| Erich Opitz (1909–1953) | Physiologist in Kiel<br>Extensive research on physiology of asphyxiation, $O_2$ deficiency, brain hypoxia |
| Joachim Rauschke (1957) | Arterial obstruction in supracervical hanging |

to the position of the rope was considered, along with damage of the larynx, congestion of inner organs, and foam around the mouth and nose. In cases of postmortem hanging, these vital signs would be missing.

In his famous book *De sedibus et causis morborum*, Giovanni Battista Morgagni (1682–1771) gives descriptions of morbid anatomical findings in strangulation (e.g. fluidity of blood in hanging, rupture of the thyrohyoid membrane and surrounding muscles in hanging, haemorrhages from the outer ear in hanging, and cyanosis and congestion of the face).

Paolo Zacchia (1584–1659) had already described some external findings in drowning, such as foam around the mouth and nose, and thought these were due to congestion of the cerebral ventricles by the water and the impaired respiration due to the forced expiration of air. In his opinion, drowning was more likely to be due to impaired respiration than to the amount of water swallowed. In contrast, Paré was of the opinion that death due to drowning was a result of swallowing too much water (Table 1.4).

In 1773 Scheele and, almost simultaneously, in 1774 Priestley discovered oxygen and the way was open for Lavoisier: 'To unravel the mystery of combustion (1776) and, in one stroke, to clarify completely the mechanism of respiration, thereby putting an end to centuries of obscurity. May I remark in passing that there was a time when arteries where conceded to be simply pipes and ensuring the distribution of air in the body' (Thomas, 1974). Unfortunately, Lavoisier's discoveries were ended by the French Revolution, which was also responsible for his untimely death.

**Table 1.4 Observations and remarks on drowning**

| Forensic scientist | Area of study |
|---|---|
| Ambroise Paré (1510–1590) | Drowning due to swallowing of too much water |
| Paolo Zacchia (1584–1659) | Foam before mouth and nose<br>Cause of death more likely due to impaired respiration than swallowing too much water |
| Arnold Paltauf (1860–1893) | Famous monograph on drowning, which is full of experimental findings and autopsy observations: *Über den Tod durch Ertrinken nach Studien an Menschen und Thieren.* Vienna, Urban & Schwarzenberg, 1888 |

Thomas summarizes Lavoisier's merits as follows:

> 'From the forensic point of view his discovery provided the key to the problem of asphyxia. It may be necessary to remind the reader that until then, death by drowning, for instance, was attributed to excessive penetration of water into the gastrointestinal tract (hence, the German word "Ertrinkung" and the Dutch expression "Verdrinking" meaning too much absorption of drink). The poor creature which was unfortunate enough to be recovered alive underwent the ordeal of enemas destined to evacuate the water from his gastrointestinal tract.'

In the second part of the 19th century, forensic medicine changed from a 'book science' to a modern scientific discipline where new knowledge was achieved by systematic observations at autopsies and experiments.

Auguste Ambroise Tardieu immortalized his name when he described subpleural petechiae, which bear his name although they had already been fully described by Bayard in 1847 in a case of infanticide, as Tardieu had reluctantly to admit (Figure 1.8a,b).

### *Tardieu's spots and asphyxia*

Subpleural, epicardial and pericranial ecchymoses have been described in cases of suffocation since the 18th century. The first description of ecchymoses is attributed to the Göttinger obstetrician Johann Georg Roederer (1726–1763). In the 19th century, great diagnostic importance was attributed to ecchymoses. Parisian-born Tardieu was the most determined defender of the specificity of ecchymoses for suffocations and he belongs to the most important specialists of forensic medicine of his time. He completed his study of medicine in Paris in 1843. After many years of lecturing, he was appointed

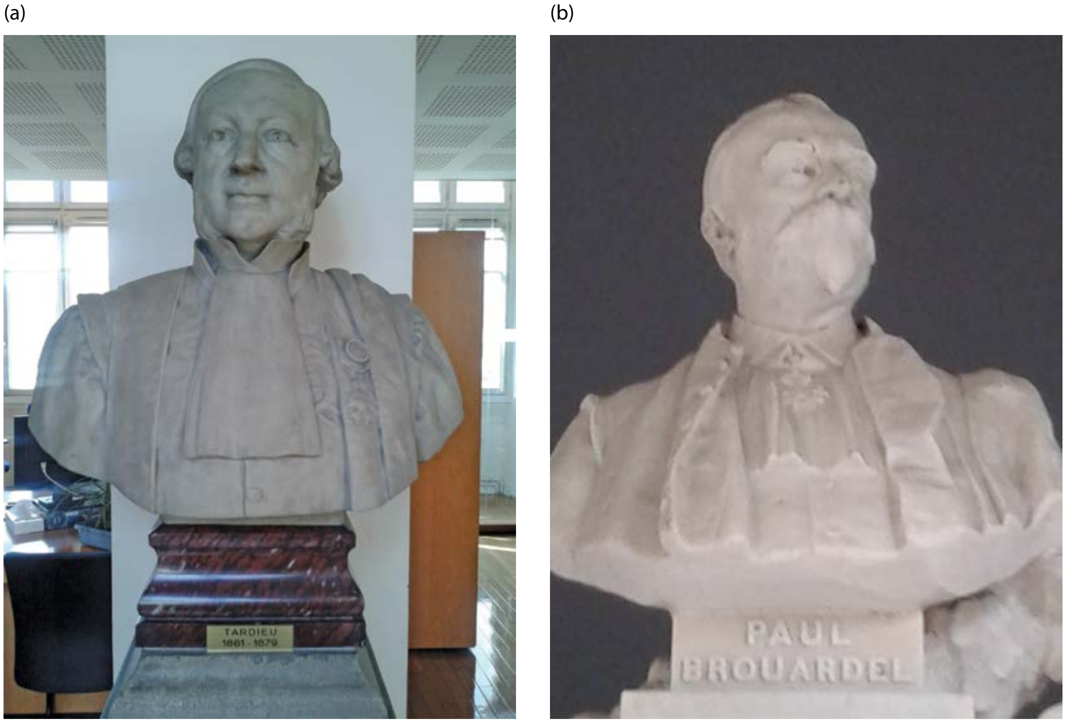


**Figure 1.8** Busts in the Institute of Forensic Medicine in Paris: (a) Auguste Ambroise Tardieu (1818–1879); (b) Paul Brouardel (1837–1906).

professor at Paris University in 1861. He thought that he could diagnose violent suffocation by means of ecchymoses and claimed that he was able to differentiate violent suffocation from other unnatural causes of death (hanging, choking, strangulation and drowning).

Other physicians, among them numerous German medical examiners, proved Tardieu's opinions wrong through observations and animal studies. The most prominent disputant against Tardieu's false doctrine was Liman. Carl Liman was born in Berlin in 1818 and died there in 1891. He studied medicine in Berlin, Bonn, Heidelberg and Halle/Saale and completed his studies in Halle/Saale in 1842. From 1861, he was an assistant at the Praktische Unterrichtsanstalt für Staatsarzneikunde in Berlin where his uncle, Johann Ludwig Casper, was Director from 1841 to 1864. After Casper's death, he was appointed Professor and Head of the Institute. With today's knowledge of pathophysiology, it is clear that haemorrhages cannot have a specificity for suffocation, let alone a specific form of suffocation. Therefore – as recommended by Geserick et al. (2005) – the confusing term of 'suffocation haemorrhages' should be avoided.

### *Eduard von Hofmann and the Austrian School of Forensic Medicine*

One of the leading experts of that time was Eduard von Hofmann who had qualified as a university lecturer in Prague and was called to the newly founded Chair for State Medicine at the University of Innsbruck in 1869. Later, in 1875, he was appointed Professor of Forensic Medicine at the University of Vienna. Von Hofmann became world famous for the classical *Lehrbuch der Gerichtlichen Medicin* (Textbook of Forensic Medicine), which was published during his lifetime between 1878 and 1897 in eight editions and was later continued by Alexander Kolisko (1903) and Albin Haberda (1919/1925 and 1927) after his death. He published widely on different kinds of asphyxia. In Innsbruck he had already published on '*Kindestötung oder unbeabsichtigte Strangulation mit der um den Hals geschlungenen Nabelschnur*' ('Infanticide or involuntary strangulation due to neck compression by the umbilical cord'). During his time in Vienna, he published extensively about hanging and notably clarified the mechanism of hanging (cerebral ischaemia). Some of his publications are listed in Table 1.5.

Von Hofmann's experiments on human bodies to demonstrate the occlusion of the neck arteries in cases of suspension were later continued by his pupil Albin Haberda (1868–1933) and Reiners. Members of the Austrian School of Forensic Medicine made a considerable contribution to the understanding of death by strangulation, among them Haberda, Arnold Paltauf (1860–1893), Fritz Reuter (1875–1959) and Walther Schwarzacher (1892–1958). In Vienna Arnold Paltauf wrote his famous book on death by drowning, which is full of observations from autopsies and experimental results. Schwarzacher carried out physical studies on traction forces in death from hanging which are still of great practical relevance today. A later Head of the Institute of Forensic Medicine at the University of Vienna, Leopold Breitenecker (1902–1981) habilitated in 1939 with

**Table 1.5** **Some of Eduard von Hofmann's publications on asphyxia**

| Date | Title of publication |
|---|---|
| 1876 | *Über den Tod durch Erhängen*<br>(About death due to hanging) |
| 1876 | *Justifikation durch den Strang*<br>(Justification by hanging) |
| 1878 | *Über Stellungen von Erhängten*<br>(About positions in hanging) |
| 1879 | Ein Fall von Selbsterdrosselung<br>(A case of suicidal ligature strangulation) |
| 1880 | *Blutung aus den Ohren bei einem Erhängten*<br>(Outer ear haemorrhages in hanging) |
| 1881 | *Zur Kenntnis der Befunde am Hals von Erhängten*<br>(Contribution on findings on the neck in hanging) |
| 1888 | *Selbstmord durch Erhängen oder Erdrosselung und Suspension durch fremde Hand*<br>(Suicide by hanging or ligature strangulation and homicidal suspension) |
| 1889 | *Über postmortale Rupturen des Sternocleidomastoideus*<br>(On postmortem ruptures of sternocleidomastoideus muscle) |

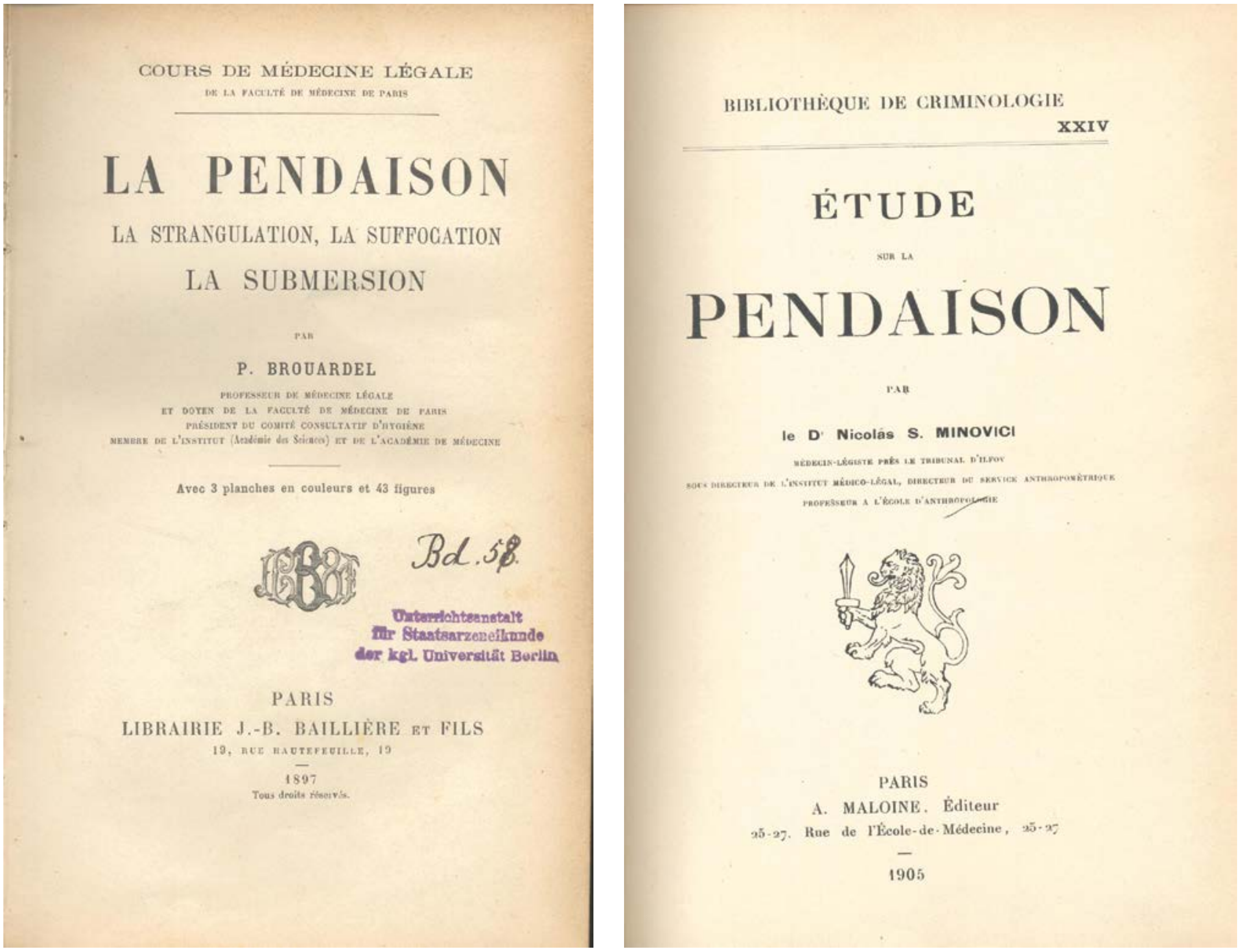
a thesis on the elimination rate of carbon monoxide from the blood of surviving victims.

### Famous books

Many famous books on asphyxiation were published in the 19th century, including those by Paul Brouardel (1897) and Nicolae Minovici (1905) (Figure 1.9a,b). These books are full of illustrations of body positions in hanging (Figure 1.10a–d). Very unusual observations were reported in these old books – for instance, on a double suicide by hanging with a single ligature – and it is therefore still worth reading them today.

### Double suicide by hanging with a single ligature

In 2017, Behera et al. published a case report of a double suicide by hanging and they thought this was a unique finding. However, in older literature such cases were described. The case is, of course, unique in the sense that a common ligature was used by the couple to complete the process of hanging. In his famous 1895 Textbook of Forensic Medicine *Lehrbuch*



(a)

COURS DE MÉDECINE LÉGALE
DE LA FACULTÉ DE MÉDECINE DE PARIS

LA PENDAISON
LA STRANGULATION, LA SUFFOCATION
LA SUBMERSION

PAR

P. BROUARDEL
PROFESSEUR DE MÉDECINE LÉGALE
ET DOYEN DE LA FACULTÉ DE MÉDECINE DE PARIS
PRÉSIDENT DU COMITÉ CONSULTATIF D'HYGIÈNE
MEMBRE DE L'INSTITUT (Académie des Sciences) ET DE L'ACADÉMIE DE MÉDECINE

Avec 3 planches en couleurs et 43 figures

Bd. 58
Unterrichtsanstalt
für Staatsarzeneikunde
der kgl. Universität Berlin

PARIS
LIBRAIRIE J.-B. BAILLIÈRE ET FILS
19, RUE HAUTEFEUILLE, 19

1897
Tous droits réservés.

(b)

BIBLIOTHÈQUE DE CRIMINOLOGIE
XXIV

ÉTUDE
SUR LA
PENDAISON

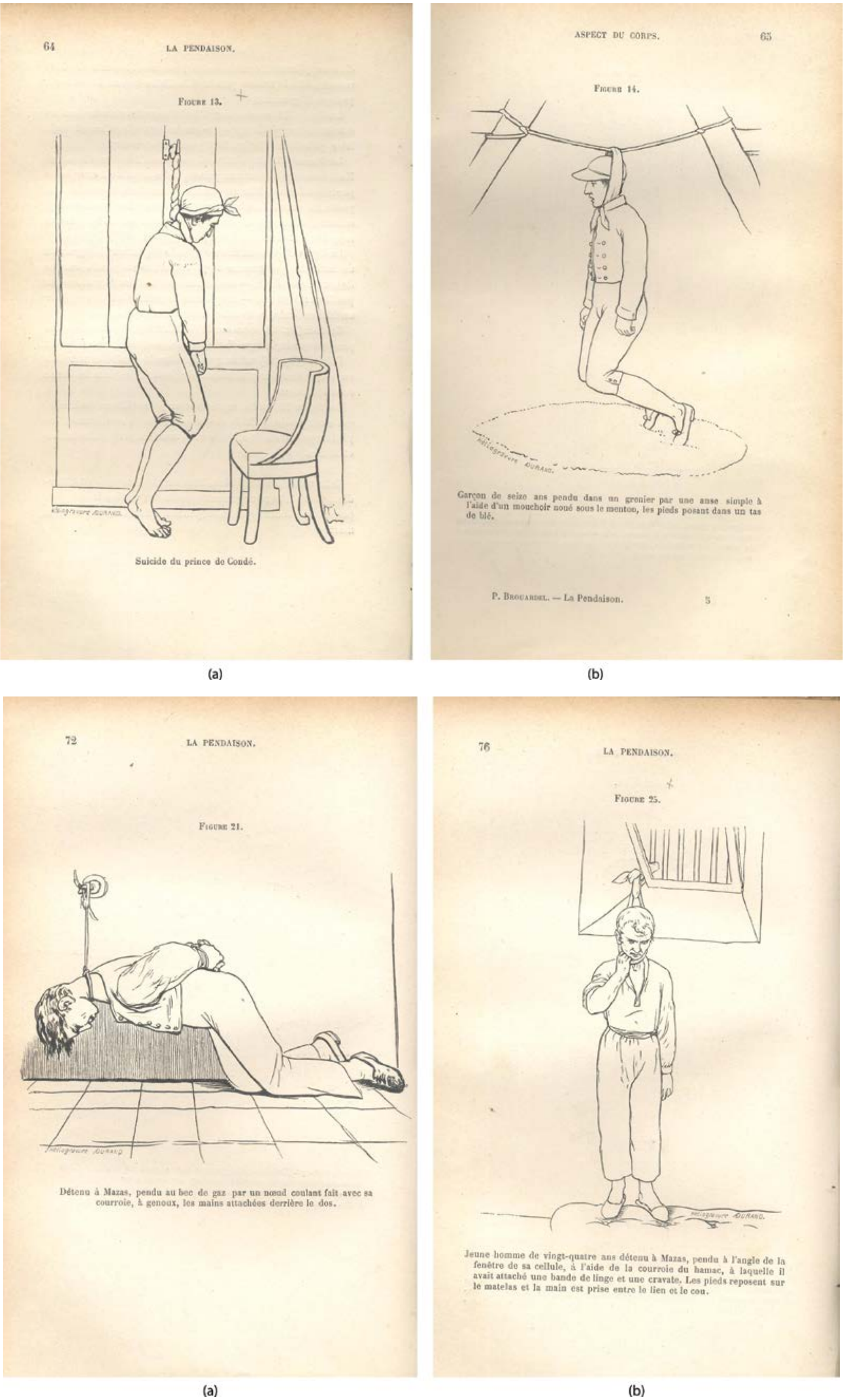
PAR

le Dr Nicolás S. MINOVICI
MÉDECIN-LÉGISTE PRÈS LE TRIBUNAL D'ILFOV
SOUS DIRECTEUR DE L'INSTITUT MÉDICO-LÉGAL, DIRECTEUR DU SERVICE ANTHROPOMÉTRIQUE
PROFESSEUR A L'ÉCOLE D'ANTHROPOLOGIE

PARIS
A. MALOINE, Éditeur
25-27, Rue de l'École-de-Médecine, 25-27

1905

**Figure 1.9** (a), (b) Title pages of books by Brouardel and Minovici.



64 LA PENDAISON.

FIGURE 13.

Suicide du prince de Condé.

ASPECT DU CORPS. 65

FIGURE 14.

Garçon de seize ans pendu dans un grenier par une anse simple à l'aide d'un mouchoir noué sous le menton, les pieds posant dans un tas de blé.

P. BROUARDEL. — La Pendaison. 5

(a)

(b)

72 LA PENDAISON.

FIGURE 21.

Détenu à Mazas, pendu au bec de gaz par un nœud coulant fait avec sa courroie, à genoux, les mains attachées derrière le dos.

76 LA PENDAISON.

FIGURE 25.

Jeune homme de vingt-quatre ans détenu à Mazas, pendu à l'angle de la fenêtre de sa cellule, à l'aide de la courroie du hamac, à laquelle il avait attaché une bande de linge et une cravate. Les pieds reposent sur le matelas et la main est prise entre le lien et le cou.

(a)

(b)

**Figure 1.10** (a)–(d) Body positions in hanging, from Brouardel P. *La pendaison, la strangulation, la suffocation, la submersion*. Paris, J.-B. Bailliere et Fils, 1897.

*der Gerichtlichen Medicin*, Eduard von Hofmann included a case of double suicide by hanging (Figure 1.11).

A 17-year-old girl and her 20-year old friend committed suicide in a hotel by hanging using one ligature. The door was open, and the ligature, a bed sheet, was running over the opened door. On the left side, the girl was hanging with the knot under the chin, and on the right side the man with the knot in the neck. A suicide note was found. According to this note, they tried to commit suicide at first by intoxication. After that failed, they committed suicide by hanging with the one ligature.

In Brouardel's 1897 textbook *La pendaison, la strangulation, la suffocation, la submersion* a very similar picture can be found. A similar case was also published by Reuter (1933) (Figure 1.12).

Even in 'unique' cases, the old Latin phrase is proved right: *'Nihil novo sub sole' ('There is nothing new under the sun.')*.

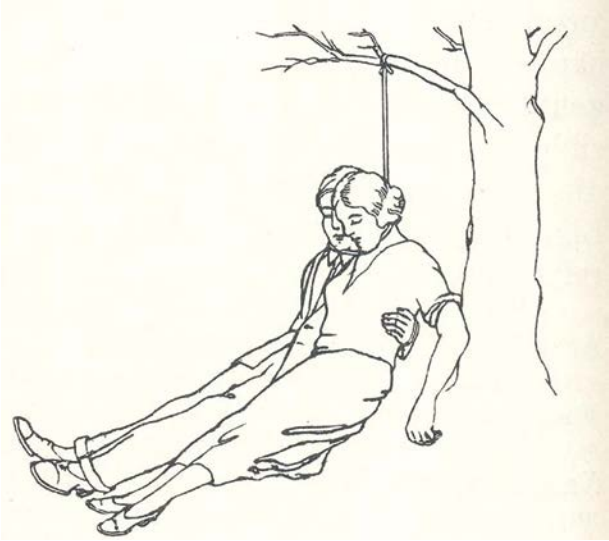


**Figure 1.12** Double suicide by hanging using one ligature (from Reuter F. *Lehrbuch der Gerichtlichen Medizin, Textbook of Forensic Medicine*, Berlin, Urban & Schwarzenberg, 1933).

## Further developments in the 19th and 20th centuries

On the 100th anniversary of the German Society of Legal Medicine a Festschrift in German as well as a commemorative issue of *Forensic Science International* were published. In both the Festschrift and the commemorative issue of *FSI*, the merits and contributions of German-speaking specialists in forensic medicine on various topics of our discipline were summarized. According to Püschel et al. (2004), who wrote the contribution on asphyxia-related death in the 19th and early 20th centuries, questions concerning the morphology and pathophysiology of asphyxial death were an almost exclusive domain of German forensic medicine, and continuous progress is still being achieved in asphyxia-related science. At the end of his contribution there is a list of the most important titles in the German language on asphyxia-related deaths. A modified version of this list is given in Table 1.6.

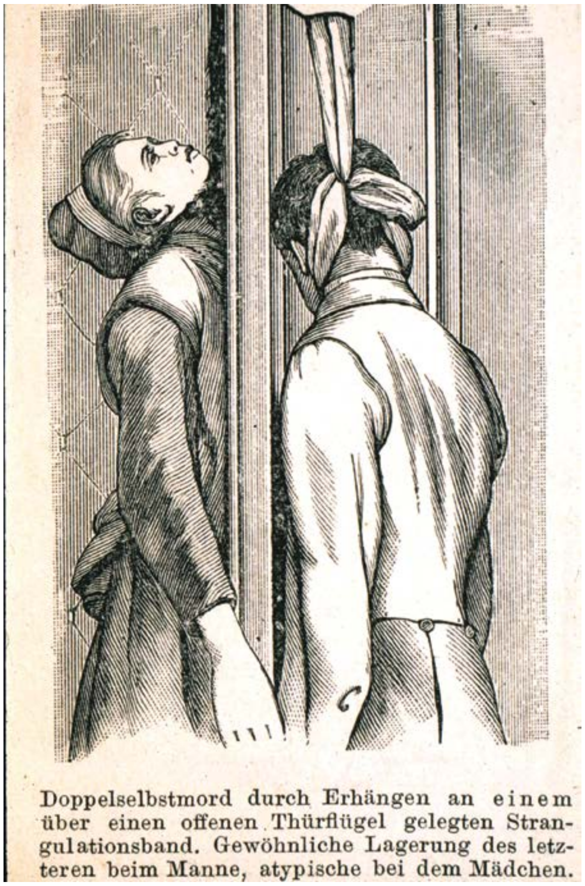

Doppelselbstmord durch Erhängen an einem über einen offenen Thürflügel gelegten Strangulationsband. Gewöhnliche Lagerung des letzteren beim Manne, atypische bei dem Mädchen.

**Figure 1.11** Double suicide by hanging using one ligature, from Von Hofmann E. *Lehrbuch der Gerichtlichen Medicin, Textbook of Forensic Medicine*, 7th ed. Vienna, Urban & Schwarzenberg, 1895.

According to records at the Institute of Legal Medicine in Hamburg, Germany, one third of all suicides, one fourth of all homicides and a significant number of all fatal accidents can be attributed to asphyxia. Püschel et al. (2004) identified typical questions addressed to the forensic pathologists by investigating officers:

- Is this a suicidal or homicidal manner of death?
- Was the victim hanged after strangling to fake a suicide?
- Could the fatality be accidental, e.g. from an autoerotic accident?
- Can a natural death be assumed in cases with suspicious death scene findings, e.g. death in connection with sexual intercourse?
- If instantaneous neurogenic cardiac arrest is diagnosed, was it the result of manual strangling after a short hit against the neck?

In the experience of Püschel et al., answering these questions may be difficult if there are no objective witnesses and if information on the deceased's history is missing.

One of the most famous trials in Germany in the 20th century was the so-called 'Hetzel case' or 'Veal rope trial'

**Table 1.6 Most important titles in the German language on asphyxia-related deaths**

| Author(s) | Date | Title of publication |
|---|---|---|
| Liman | 1868 | *Bemerkungen zum Tod durch Ersticken, Erdrosseln und Erwürgen*<br>(Remarks on death by asphyxiation, ligature strangulation and throttling) |
| Ecker | 1870 | *Die Stellung des weichen Gaumens beim Tod durch Erhängen*<br>(On the position of the palate in death by hanging) |
| von Hofmann | 1880 | *Blutungen aus den Ohren eines Erhängten nebst Mitteilungen über analoge Befunde*<br>(Haemorrhages from the ear in hanging and remarks on analogues findings) |
| Lesser | 1880 | *Zur Beurteilung des diagnostischen Wertes der Strangmarke in Bezug auf die Frage: Ist das Erhängen intra vitam oder post mortem erfolgt?*<br>(Assessment of the diagnostic value of the strangulation mark concerning the question: Was the hanging intra vitam or post mortem?) |
| Langreuter | 1886 | *Über die mechanischen Verhältnisse des Strangulationstodes*<br>(About the mechanical relations in death by strangulation) |
| Straßmann F | 1887 | *Einiges über das Aufhängen von Leichen*<br>(Some remarks about the hanging of bodies) |
| Binner | 1888 | *Ein Fall von Selbstmord durch Erwürgen*<br>(A case of suicide by throttling) |
| Haberda, Reiner | 1894 | *Experimentelle und kritische Beiträge zur Lehre vom Tod durch Erhängen*<br>(Experimental and critical contribution about the doctrine of death by hanging) |
| Kratter | 1895 | *Zur Diagnose der Erstickung*<br>(On the diagnosis of asphyxiation) |
| Reineboth | 1895 | *Tod eines Tracheotomierten durch Erhängen*<br>(Death by hanging in a case of tracheostoma) |
| Schultz | 1898 | *Über vitale und postmortale Strangulation*<br>(About vital and postmortem strangulation) |
| Puppe | 1907 | *Die Diagnose der Erstickung durch weiche Bedeckungen*<br>(On the diagnosis of asphyxiation by soft coverings) |
| Meixner | 1919 | *Fragliches Erhängen bei Lage der Schlinge über dem Kinn*<br>(Doubtful hanging by position of the rope over the chin) |
| Reuter | 1922 | *Über das Vorkommen, die Entstehung und die Bedeutung von Muskelblutungen beim Erstickungstode*<br>(On the occurrence, origin and importance of muscular haemorrhages in asphyxia-related deaths) |
| Straßmann G | 1924 | *Der Verschluss der Atemwege beim Erhängen und Erdrosseln*<br>(On the occlusion of the respiratory tract in hanging and ligature strangulation) |
| Ziemke | 1925 | *Über zufälliges Erhängen und seine Beziehungen zu sexuellen Perversitäten*<br>(Accidental hanging and its relation to sexual perversity) |
| Werkgartner | 1926 | *Tötung der Ehefrau durch Erwürgen und Vortäuschen eines Selbstmordes durch Erhängen*<br>(Homicide of spouse by throttling and fake of a suicide by hanging) |
| Hering | 1927 | *Die Carotissinusreflexe*<br>(Carotis sinus relfexes) |
| Schwarzacher | 1928 | *Beiträge zum Mechanismus des Erhängungstodes*<br>(Contributions to the mechanics of death by hanging) |
| Lochte | 1930 | *Über einen Fall von Tod durch Erdrosseln und über die Bedeutung des Sinus caroticus (Hering)*<br>(About a case of death by ligature strangulation and the importance of the carotic sinus (Hering)) |
| Fraenckel | 1930 | *Seltene Strangulierungsarten beim Selbstmord*<br>(About rare suicidal kinds of strangulation) |
| Esser | 1933 | *Zur Frage des Erwürgens ohne lokale anatomische Spuren beim Menschen und im Tierversuch*<br>(On the question of throttling without local anatomical findings in humans and animal experiments) |
| Kalle | 1933 | *Beobachtungen über den Tod bei Hinrichtungen mit dem Strang*<br>(Observations in cases of excecution with the rope) |
| Schrader | 1937 | *Neuere Wege in der Diagnose der gewaltsamen Erstickung*<br>(New directions in the diagnosis in mechanical asphyxia) |
| Fritz | 1940 | *Selbstmord oder Unfall? Ein ungewöhnlicher Fall von Erhängen durch den Halsausschnitt eines Anstaltskleides*<br>(Suicide or accident? An unusual case of hanging by the neckline of a mental-home dress) |
| Walcher | 1943 | *Über Erstickung*<br>(About asphyxiation) |
| Büchner | 1944 | *Die pathogenetische Bedeutung des allgemeinen Sauerstoffmangels*<br>(On the pathogenetic importance of oxygen deficiency) |
| Berg | 1952 | *Eine für Erhängung charakteristische vitale Reaktion*<br>(About a characteristic vital reaction in cases of hanging) |

(*Continued*)

**Table 1.6 (*Continued*)** **Most important titles in the German language on asphyxia-related deaths**

| Author(s) | Date | Title of publication |
|---|---|---|
| Frei | 1955 | *Beitrag zur Spurenkunde des Suizids durch Erhängen und Erdrosseln*<br>(Contribution on the stain analysis of suicide by hanging and ligature strangulation) |
| Mueller | 1961 | *Tierexperimentelle Studien über den Erstickungstod, insbesondere über Erdrosseln und Erwürgen*<br>(Animal experimental study on asphyxiation especially ligature strangulation and throttling) |
| Janssen | 1963 | *Riesenzellbildung bei Erstickung*<br>(Pulmonary giant cells in asphyxial death) |
| Holczabek | 1964 | *Erstaunliche Aktionsfähigkeit nach Erhängungsversuch mit Reißen des Strickes*<br>(Astonishing physical activity in a case of attempting hanging with rupture of the rope) |
| Simon | 1968 | *Vitale Reaktionen im Bereich der Lendenwirbelsäule beim Erhängen*<br>(Vital reactions of the lumbar spine in cases of hanging) |
| Prokop | 1970 | *Der Fall Hetzel*<br>(The Hetzel case) |
| Jarosch | 1971 | *Experimentelle Untersuchungen über vasculäre und neurale Mechanismen bei gewaltsamer Erstickung*<br>(Experimental investigations about vascular and neural mechanisms in mechanical asphyxiation) |
| Althoff | 1975 | *Untersuchungen über den Aussagewert von Mikrospuren beim Tod durch Strangulation*<br>(Investigations about the importance of micro-stains in death by strangulation) |
| Bonte | 1975 | *Mehrfachverknotungen bei Selbstmord durch Erdrosseln*<br>(Several knottings in suicide by ligature strangulation) |
| Bratzke | 1975 | *Erhängen oder Drosseln? – Tablettenvergiftung*<br>(Hanging or throttling? – Intoxication by pills) |
| Pollak | 1975 | *Über die Häufigkeit des Lungenödems beim Erhängungstod*<br>(About the frequency of pulmonary oedema in cases of hanging) |
| Gerchow | 1976 | *Zur Frage der Handlungsfähigkeit bei mechanischer Erstickung*<br>(On the question of physical acitivity in mechanical asphyxiation) |
| Saternus | 1977 | *Verletzungen des Halses durch direkte und indirekte Gewalteinwirkungen*<br>(Injuries of the neck by direct and indirect mechanical injury) |
| Brinkmann | 1978 | *Vitale Reaktionen in der Lungenstrombahn bei Tod durch Strangulation*<br>(Vital reactions in the pulmonary vessels in cases of death by strangulation) |
| Adebahr | 1981 | *Über die Wertigkeit von Zyanose und Petechien bei Gewalteinwirkung auf den Hals und gleichzeitig vorhandener erheblicher Anämie*<br>(On the importance of cyanosis and petechiae in cases of neck compression and simultaneous advanced anaemia) |
| Brinkmann et al. | 1981 | *Zur Pathophysiologie der Atmung und des Kreislaufs bei Tod durch obstruktive Asphyxie*<br>(On the pathophysiology of respiration and circulation in death by obstructive asphyxia) |
| Kleiner et al. | 1982 | *Zur Pathologie des Erhängens unter besonderer Berücksichtigung vitaler Reaktionen*<br>(On the pathology of hanging with special reference to vital reactions) |
| Püschel | 1982 | *Vitale Reaktionen zum Beweis des Todes durch Strangulation*<br>(Vital reactions as proof of death by strangulation) |
| Madea | 1985 | *Erdrosseln – Mord oder Selbstmord*<br>(Ligature strangulation – homicide or suicide) |
| Maxeiner | 1986 | *Anleitung für eine vollständige Kehlkopfpräparation*<br>(Guidance of a complete preparation of the throat skeleton) |
| Madea et al. | 1987 | *Zur Ausbildung der Strangmarke bei Wasserexposition der Leiche*<br>(Influence of water exposure on the appearance of strangulation marks) |
| Eisenmenger et al. | 1990 | *Pathobiochemische Aspekte der obstruktiven Asphyxie – eine Bestandsaufnahme*<br>(Pathobiochemical aspects of obstructive asphyxia – state of the art) |
| Geserick | 1990 | *Zur Bedeutung von Stauungsblutungen bei der gewaltsamen Asphyxie*<br>(On the importance of petechial haemorrhages in mechanical asphyxia) |
| Kauert | 1990 | *Zur Frage der strangulationsbedingten craniokaudalen Konzentrationsdifferenz biochemischer Parameter am Beispiel der Katecholamine*<br>(On the question of craniocaudal concentration differences of biochemical parameters in strangulation on the example of catecholamines) |
| Madea | 1990/ 1993 | Death in a head-down position |
| Betz et al. | 1994 | Pulmonary giant cells and traumatic asphyxia |
| Grellner, Madea | 1996 | Immunohistochemical characterization of alveolar macrophages and pulmonary giant cells in fatal asphyxia |
| Doberentz et al. | 2011 | Histological examination of the carotid bifurcation in a case of violence against the neck |

*Source:* Modified from Püschel K, Türk E, Lach H. *Forensic Sci Int* 2004;**144:**211–214.

(*Kälberstrick trial)* (see Preface). A young woman was found dead in a road ditch (Figure 1.13a). There was a dried mark on the front of her neck (Figure 1.13b,c). Based on the autopsy report and the photographs, the diagnosis of ligature strangulation as cause of death was made and the suspect was sentenced to long life imprisonment. Later, during the hearing at a retrial, it was claimed that the dried abrasion of the neck was due not to ligature strangulation but to the skin lying on a stick in the ditch. The abrasion on the front of the neck was a postmortem abrasion. However, abrasions or dryings of the skin of the face were missing and the young woman had both petechial haemorrhages and haemorrhages of the neck. Obviously, the acquittal after the new hearing retrial was a mistake.

## Definitions and classification of asphyxia

### Defining asphyxia

According to Püschel et al. (2004), there are almost as many different ways to classify asphyxial death as there are authors, and there are numerous definitions of the term 'asphyxia' itself, too. Asphyxia is a failure of body cells to either receive or utilize oxygen. This is accompanied by an increase in the blood carbon dioxide level. This has led to the differentiation between 'external' and 'internal' asphyxia. In the medicolegal context, asphyxia refers mainly to forms of external hypoxia and it can be further divided into mechanical and environmental asphyxia. Of greatest importance are fatalities due to mechanical asphyxia. Environmental asphyxia covers special sets of circumstances, such as entrapment in an airtight enclosure.

### Classification of asphyxiation

Several different classifications of asphyxiation have been used in the past. The term 'asphyxiation' derives from the Greek 'asphyxia', which means pulselessness. In physiological terms, asphyxiation is defined as hypoxaemia accompanied by hypercapnia. The German physiologist Opitz (1909–1953) differentiated external from internal asphyxiation (Opitz, 1950). 'External' asphyxiation by gagging, drowning, neck compression or strangulation is mainly asphyctic asphyxiation while 'internal' asphyxiation is mainly hypoxic asphyxiation (Figure 1.14). Different pathomechanisms of hypoxidosis are shown in Figure 1.15. Subgroups of 'external' asphyxia are shown in Figure 1.16 and Table 1.7. A flow chart of different types of asphyxiation was developed by Byard (2017b) (Figure 1.17). The main pathomechanisms leading to death in different types of strangulation are shown in Figure 1.18. While in hanging, especially suspended hanging, cerebral ischaemia is the leading pathomechanism, in throttling the airway compression may be predominant.

A further classification of rapid anoxial death is shown in Table 1.8. Definitions of terms in the proposed unified classification of asphyxia death by Sauvageau and Geberth (2013) can be found in Table 1.9. Byard (2017b) has recommended definitions and terms for drowning and subclasses of drowning (Table 1.10).

## General signs of asphyxia

Certain general signs of asphyxia (congestion, cyanosis, fluidity of blood, dilatation of the right side of the heart and petechiae) were described in the classical textbooks of forensic medicine of the 19th century. The general signs of asphyxia now recognized include cyanosis, fluidity of cadaveric blood, subpleural and subepicardial ecchymosis, petechial haemorrhages, and anaemia of the spleen. However, these signs of asphyxia are to a certain extent signs that 'all causes of death have in common' and the diagnosis of asphyxia 'does not tell much except that the examined person is dead' (Puppe, 1907).

Walcher (1943) stated,

> 'The history of research into anatomical findings in fatal asphyxia is to a great extent the history of misconceptions. Nobody who knows the articles that have been published on vital findings in asphyxia can help getting this impression.'

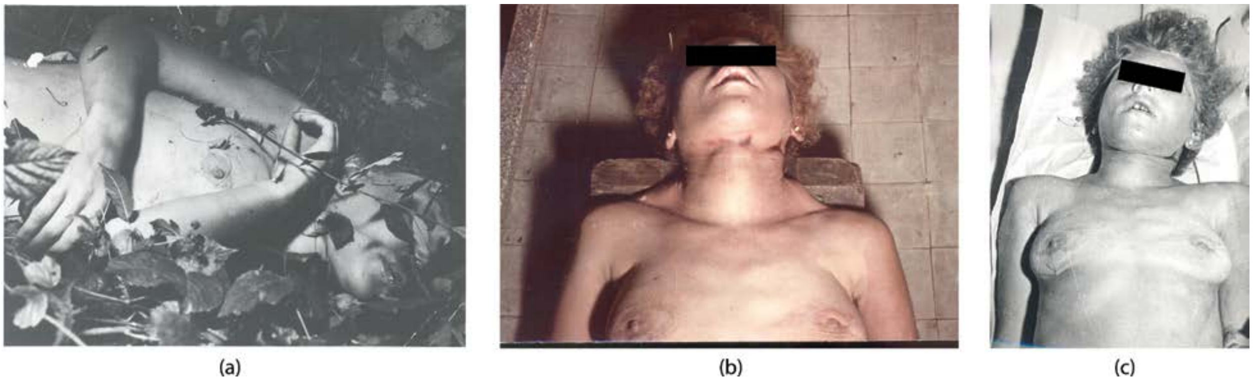


**Figure 1.13** The so-called 'Hetzel case' (veal rope trial). (a) A young woman was found naked in a road ditch. (b), (c) Obvious strangulation marks on the front of the neck (probably ligature strangulation). Later in a retrial postmortem origin of the strangulation mark was claimed.

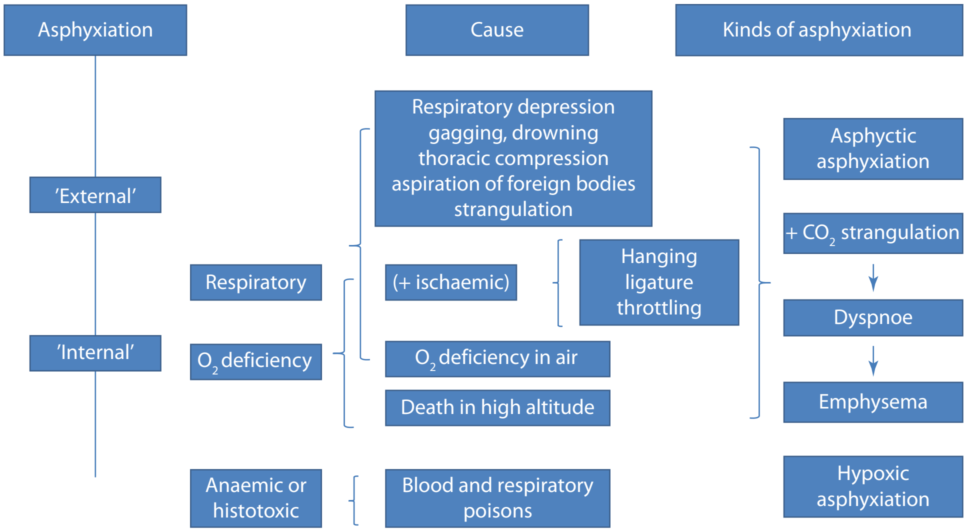

Asphyxiation
Cause
Kinds of asphyxiation
'External'
'Internal'
Respiratory
$O_2$ deficiency
Anaemic or histotoxic
Respiratory depression gagging, drowning thoracic compression aspiration of foreign bodies strangulation
(+ ischaemic)
Hanging ligature throttling
$O_2$ deficiency in air
Death in high altitude
Blood and respiratory poisons
Asphyctic asphyxiation
+ $CO_2$ strangulation
Dyspnoe
Emphysema
Hypoxic asphyxiation

**Figure 1.14** Classification of asphyxiation (according to Opitz, 1950).

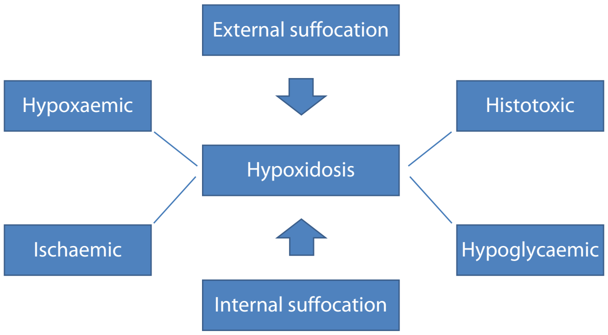

External suffocation
Hypoxaemic
Histotoxic
Hypoxidosis
Ischaemic
Hypoglycaemic
Internal suffocation

**Figure 1.15** Pathomechanisms of hypoxidosis (according to Brinkmann et al., 2004).

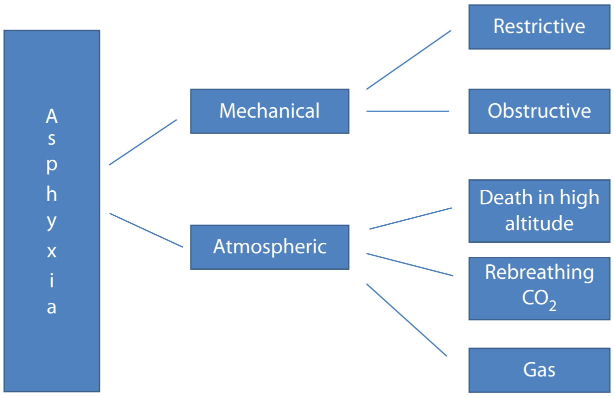

Asphyxia
Mechanical
Atmospheric
Restrictive
Obstructive
Death in high altitude
Rebreathing $CO_2$
Gas

**Figure 1.16** Subgroups of 'external' asphyxia (according to Brinkmann et al., 2004).

**Table 1.7 Subgroup of 'external' asphyxia (restrictive and obstructive asphyxia)**

| External asphyxia | Cause |
|---|---|
| Restrictive | Haemato-, pneumo- and hydrothorax<br>Unstable thorax<br>Paralysis of respiratory muscles<br>Chest compression |
| Obstructive | Smothering<br>Gagging<br>Bolus death<br>Strangulation<br>Aspiration |

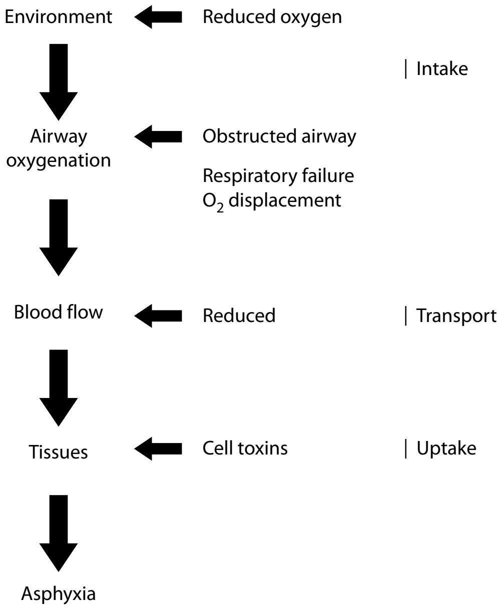

Environment
Reduced oxygen
Intake
Airway oxygenation
Obstructed airway
Respiratory failure
$O_2$ displacement
Blood flow
Reduced
Transport
Tissues
Cell toxins
Uptake
Asphyxia

**Figure 1.17** Flow chart of different types of asphyxiation (intake, transport and uptake of oxygen) (Byard RW. & Cains G. Lethal asphyxia: pathology and problems. (Reprinted by permission of Edizioni Minerva Medica from Minerva Medicolegale 2007; **127**(**4**):273–82.)

According to Berg (1984), the diagnosis of fatal asphyxia requires proof of the asphyxiating agent or traces of the asphyxiating agent on the body (e.g. rope in hanging, strangulation mark, abrasions and haemorrhages of the skin of the neck). Since the macro-morphological signs

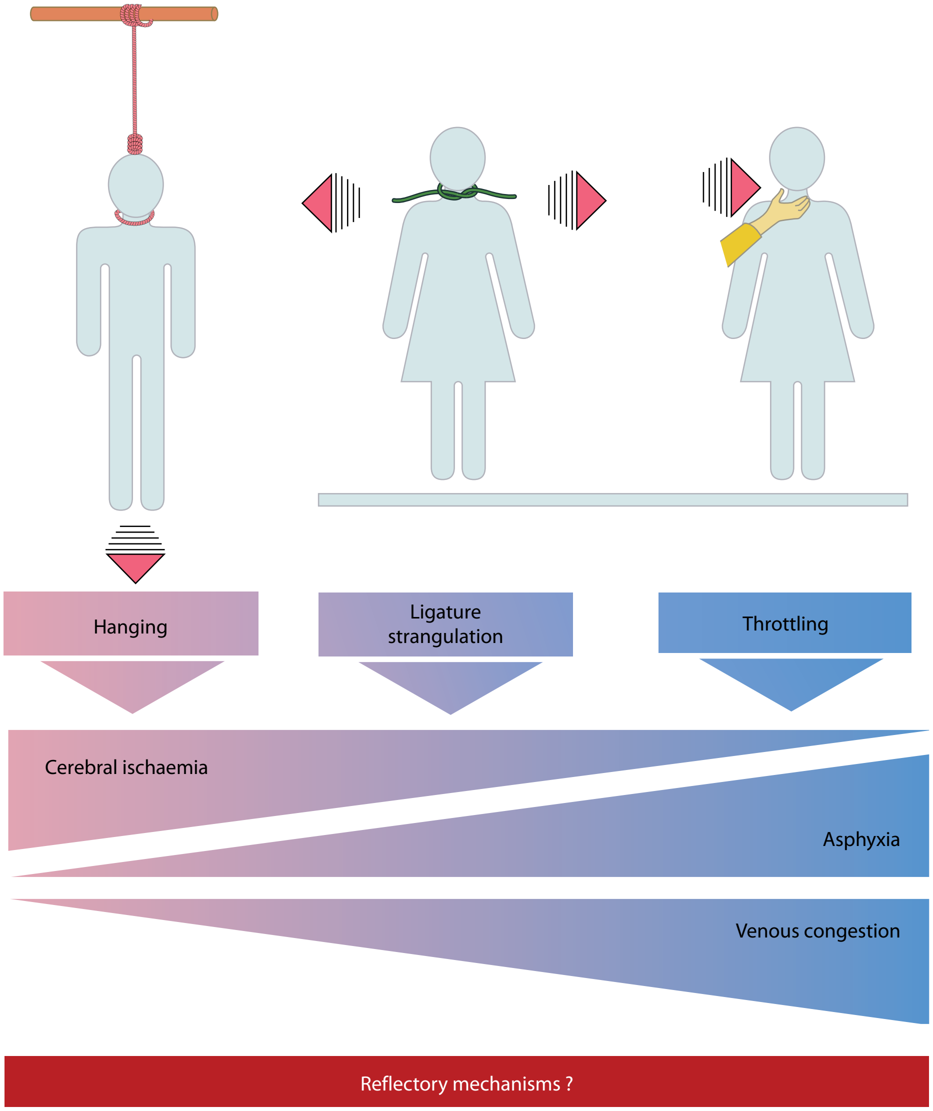


**Figure 1.18** Main pathomechanisms leading to death in different types of strangulation (modified according to Püschel K. *Vitale Reaktionen zum Beweis des Todes durch Strangulation*. Univ. Hamburg, Habilschrift, 1982 and Maxeiner, 2003).

of asphyxia may be weak or even absent at autopsy, the number of unrecorded cases of homicide by asphyxia is a great problem. Even in clear cases of homicide by asphyxia, such as by ligature strangulation (Figure 1.19a–d), a natural manner of death can be certified.

## Etiology and pathophysiology of strangulation

The main mechanisms of mechanical asphyxia are hanging, ligature strangulation and manual strangulation, or through obstruction of the airways, for instance by a soft covering such as a pillow, and thoracic compression. Death by thoracic compression may occur accidentally but also occurs in homicides. For example, the 19th-century murderers Burke and Hare killed their victims by chest compression using their body weight and obstructing the airways with their hands. They sold the bodies for dissection at anatomy lectures. This kind of killing was historically known as 'burking'.

The main mechanisms leading to death in strangulation (hanging, ligature strangulation and throttling) are compression of the neck arteries and airway obstruction. These mechanisms of death were extensively studied in the 19th century (Table 1.11).

### Compression of neck arteries

Eduard von Hofmann was the founder of a very important school of forensic medicine in Vienna. In 1876 von Hofmann held a lecture in front of the Association of Physicians of Lower Austria which pointed the way ahead when he suggested that the mechanism of death in strangulation deaths was the compression of the neck arteries. This view was confirmed in research experiments by von Hofmann himself and his pupils Haberda and Reiner.

**Table 1.8 Classification of rapid anoxial death**

| Mechanism | Classical terminology | Examples |
|---|---|---|
| Reduced oxygen tension in the respirable atmosphere | Environmental hypoxia | Rebreathing within a plastic bag sealed overhead |
| Obstruction of the mouth and nose | Upper airway obstruction | Choking; gagging; suffocation/smothering |
| Interference with the mechanics of breathing or chest compression | Positional asphyxia | Heavy load compressing chest wall and limiting respiratory excursion; acute ethanol intoxication with fall into posture causing hyperflexion of the neck; hogtying in prone position |
| Neck compression | Pressure on the neck | Strangulation; hanging; neck holds |
| Chemical inhibition of oxygen utilization for metabolism | Chemical anoxia | Cyanide and carbon monoxide poisoning |

*Source:* From Pollanen MS. *Asphyxia*, with permission from John Wiley & Sons, 2009, p225.

Since then, a great many articles on arterial compression in strangulation have been published. It is now more or less generally accepted that a minimum weight of 5 kg is needed for the carotid artery and 35 kg is needed for the vertebral artery to achieve complete compression (Tables 1.12 and 1.13). In different hanging positions (Figure 1.20a,b) a proportion of body weight is enough to occlude neck structures (Table 1.13). Walter Schwarzacher (1928) carried out physical studies on traction forces in death from hanging (Figure 1.21). For cases of supracervical hanging, Rauschke (1957) carried out experiments to measure the forces necessary to cause a complete arterial compression (Figure 1.22, and see also Figure 1.23).

**Table 1.10 Definition of terms in drowning and near-drowning (according to Byard, 2017b)**

| Term | Definition |
|---|---|
| Drowning | Death occurring within 24 hours of a submersion incident<br>Dying through submersion in and inhalation of water<br>Death from suffocation as a result of exclusion of air from the lungs by fluid, usually water<br>Suffocation and death resulting from filling of the lungs with water or other substance<br>Asphyxiation caused by submersion in a liquid<br>Death from suffocation resulting from aspiration of water or other substance or fluid<br>Process of experiencing respiratory impairment from submersion/immersion in liquid<br>Death due to asphyxia caused by submersion in fluid |
| Immersion | Immersion of body into fluid without head |
| Submersion | Immersion of body into fluid with head |
| Drowning | Lethal submersion |
| Near-drowning | Initial survival at least beyond 24 hours of an individual after suffocation due to submersion in fluid |
| Wet-drowning | Drowning with proof of fluid aspiration |
| Dry-drowning | Drowning without proof of fluid aspiration |

## Airway obstruction

In the second half of the 19th century, the pressure necessary for airway obstruction in strangulation had also been studied. In 1870, Ecker proved the airway obstruction in strangling when he sawed the frozen dead body of a hanged individual to show how the tongue was pressed against the posterior pharynx wall (Figure 1.24). However, at the end of the 19th century, the significance of this airway obstruction for fatal outcomes in hanging was already relativized when hanging deaths were observed

**Table 1.9 Definition of terms in the proposed modified classification***

| Term | Definition |
|---|---|
| Suffocation | A broad term encompassing different types of asphyxia, such as vitiated atmosphere and smothering, associated with deprivation of oxygen |
| Smothering | Asphyxia by obstruction of the air passages above the epiglottis, including the nose, mouth and pharynx |
| Choking | Asphyxia by obstruction of the air passage below the epiglottis |
| Confined space/entrapment/vitiated atmosphere | Asphyxia in an inadequate atmosphere by reduction of oxygen, displacement of oxygen by other gases, or by gases causing chemical interference with the oxygen uptake and utilization |
| Strangulation | Asphyxia by closure of the blood vessels or air passage of the neck as a result of external pressure on the neck |
| Hanging | A form of strangulation in which the pressure on the neck is applied by a constricting band tightened by the gravitational weight of the body or part of the body |
| Ligature strangulation | A form of strangulation in which the pressure on the neck is applied by a constricting band tightened by a force other than the body weight |
| Manual strangulation | A form of strangulation caused by an external pressure on the structures of the neck by hands, forearms or other limbs |
| Positional or postural asphyxia | A type of asphyxia in which the position of an individual compromises the ability to breathe |
| Traumatic asphyxia | A type of asphyxia caused by external chest compression by a heavy object |
| Drowning | Asphyxia by immersion in a liquid |
| Asphyxiation | Lack of pulse (Greek)<br>In terms of physiology, hypoxaemia accompanied by hypercapnia |

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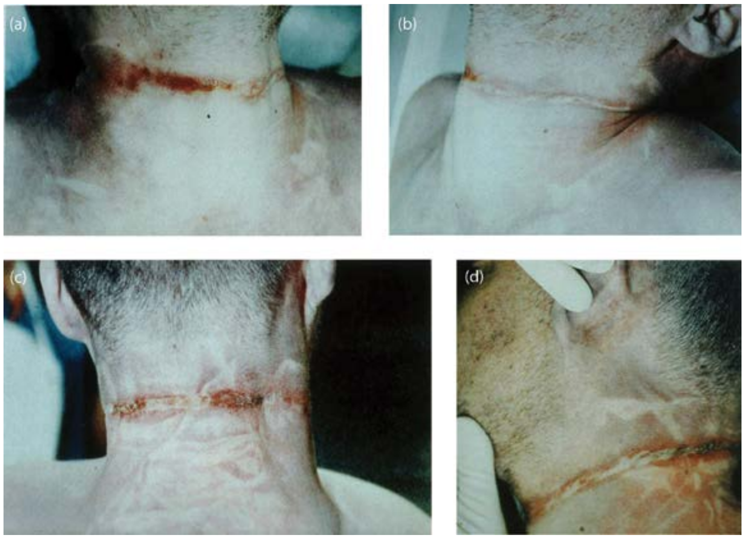

**Figure 1.19** (a)–(d) Ligature mark running horizontally around the neck. A natural manner of death had originally been certified. The ligature mark was discovered at the second external examination, which is compulsory in Germany before cremation.

**Table 1.11 Aetiology and pathophysiology of strangulation**

**Compression of neck arteries**

- von Hofmann E. *Über den Tod durch Erhängen.* (About death by hanging.) Mitteilungen des Vereins der Aerzte in Nieder-Oesterreich. II. 1876a;**8:**141–147.
- Haberda A, Reiner M. Experimentelle und kritische Beiträge zur Lehre vom Tod durch Erhängen. (Experimental and critical contributions about death by hanging.) *Vjschr Gerichtl Med* (3. Folge) 1894;**8**(Suppl.):126–147.
- Haberda A, Reiner M. Über die Ursache des raschen Eintritts der Bewußtlosigkeit bei Erhängten. (About the cause of rapid loss of consciousness in hanging.) *Vjschr Gerichtl Med* (3. Folge) 1897;**13**:155–158.
- Rauschke J. Über den Eintritt der Bewußtlosigkeit bei atypischer Erhängung. (About the angle of unconsciousness in atypical hanging.) *Dtsch Z Gesamte Gerichtl Med* 1957;**46:**206–211.
- Brinkmann B, Koops E, Wischhusen F, Kleiber M. Halskompression und arterielle Obstruktion. (Neck compression and arterial obstruction.) *Z Rechtsmed* 1981;**87:**59–73.

**Airway obstruction**

- Ecker. Die Stellung des weichen Gaumens beim Tode durch Erhängen. (On the position of the palate in death by hanging.) *Virchows Arch Anat* 1870;**49:**290–291.
- Reineboth. Tod eines Tracheotomierten durch Erhängen. (Death by hanging in a case with tracheostoma). *Vjschr Gerichtl Med* (3. Folge) 1895;**9:**265–284.
- Langreuter. Über die mechanischen Verhältnisse des Strangulationstodes. (About mechanical conditions in strangulation death.) *Vjschr Gerichtl Med* (Neue Folge) 1886;**45:**295–309.
- Straßmann G. Zum Mechanismus des Erhängungstodes. (About the mechanics of death by hanging.) *Dtsch Z Gesamte Gerichtl Med* 1922;**1:**686–694.
- Straßmann G. Der Verschluß der Atemwege beim Erhängen und Erdrosseln. (Blocking of air passages in hanging and ligature strangulation.) *Dtsch Z Gesamte Gerichtl Med* 1924;**4:**165–172.

**Carotis sinus stimulation**

- Hering HE. *Die Carotissinusreflexe*. (Carotid sinus reflex.) Dresden, Steinkopf, 1927.

**Self-experiments in strangulation**

- Minovici N. *Study on hanging.* Bucuresci, Atelierele grafice IV, Socecu str. Berzei 59, 1904/1905, pp I–V, 70, 72, 60–83, 203–207.
- Opitz E. Self experiments with ligature strangulation of the cervical arteries using a blood pressure cuff. In: Ponsold A (ed.) *Lehrbuch der Gerichtlichen Medizin.* Stuttgart, Thieme, 1950.

**Pathophysiology of neck compression**

*Heart frequency profile in cases of hanging*

- Miloslavich E. Zur Lehre vom Erhängungstode. (On death by hanging.) *Vjschr Gerichtl Med* (3. Folge) 1919;**58:**162–168.
- Kalle E. Beobachtungen über den Tod bei Hinrichtungen mit dem Strang. (Observation on death by hanging with a rope.) *Dtsch Z Gesamte Gerichtl Med* 1933;**22:**192–203.

*Pathophysiology of peracute brain ischaemia in ligature strangulation using a blood pressure cuff*

- Opitz E. Physiologie der Erstickung und des Sauerstoffmangels. (Physiology of asphyxiation and oxygen deficiency.) In: Ponsold A (ed.) *Lehrbuch der Gerichtlichen Medizin.* (Textbook of Forensic Medicine.) Stuttgart, Thieme, 1950, pp 174–218.

*Haemodynamic dysregulation in occlusion of breathing orifices*

- Swann HG, Brucer M. The cardiorespiratory and biochemical events during rapid anoxic death. V. Obstructive asphyxia. *Tex Rep Biol Med* 1949;**7:**593–603.

**Table 1.12** Pressure necessary to occlude the neck structures*

| Structure | Pressure required for occlusion |
|---|---|
| Jugular veins | 2 kg (4.5 lb) |
| Carotid arteries | 5 kg (11 lb) |
| Trachea | 15 kg (33 lb) |
| Vertebral arteries | 30 kg (66 lb) |

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**Table 1.13** Proportion of the body weight applied to the hanging ligature in incomplete hanging*

| Position of the incomplete hanging | Proportion of the body weight applied to the ligature (%) |
|---|---|
| Standing, toes touching the ground | 98 |
| Standing, feet flat on the ground | 66 |
| Kneeling, buttocks down | 74 |
| Kneeling, buttocks up | 64 |
| Sitting, back suspended upright | 18 |
| Sitting, back suspended backward | 32 |
| Lying down, face down | 18 |
| Lying down, face up | 10 |

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in individuals who had undergone tracheotomy. This finding was verified by experiments on rabbits (Reineboth, 1895). In 1886, Langreuter used a special preparation method to show that, if the thumb and index finger are placed on either side of the thyroid cartilage in manual strangulation, only a 'very low pressure' is sufficient for complete compression of the larynx. He could also confirm that, in hanging, the tongue and epiglottis are pressed against the posterior pharynx wall. Further experiments were carried out by Georg Straßmann, the son of German chemist Fritz Straßmann (1858–1940). He demonstrated a complete airway obstruction and showed that the point of suspension was in the posterior neck region or behind one ear – even in cases of incomplete suspension (Straßmann, 1924). More recent experiments showed that an average pressure of 8–12 kg is necessary to achieve complete airway obstruction in manual strangulation of an adult (see also Table 1.12).

The different mechanisms leading to death by hanging (i.e. occlusion of arteries and airway obstruction) were also well illustrated in the textbook of Albert Ponsold (1950) (Figure 1.25).

## Spinal cord/brainstem injuries

It is a common misconception that, especially in hanging fractures of the spine, consequent brainstem/spinal cord lesions cause immediate death. Of course, injuries of the cervical spinal cord can occur in cases of hanging, but they normally occur only in cases of falls from height into the noose. Researchers at the Hamburg Institute of Legal Medicine over a 10-year period found fractures of the cervical spine in only 6 out of 821 hanging deaths. These fractures were localized between segments C5 and C6. In falls from great height into the noose or vehicle-assisted suicides, the applied forces may lead to a complete decapitation.

## Carotid sinus stimulation

The German physiologist Heinrich Ewald Hering (1866–1948) published his book on carotid sinus reflex in 1927. He performed basic animal experiments but checked the results of his experimental findings in only a few experiments on humans (e.g. on one of his assistants and during an operation on the neck). He asserted that, in theory, the carotid sinus may be affected in cases of strangulation, but he carried out no experiments. In cases of hanging, the pressure on the carotid sinus may release a carotid sinus reflex with a decrease of heart frequency and blood pressure. This may induce rapid loss of consciousness. In cases of throttling, an influence on the carotid sinus also has to be taken into consideration. In cases of arteriosclerosis of the carotid sinus, light pressure would be enough to induce unconsciousness.

As far as we can see, the first time the carotid sinus reflex was significant in court was in the case of a man named von Dielingen. Von Dielingen was accused of

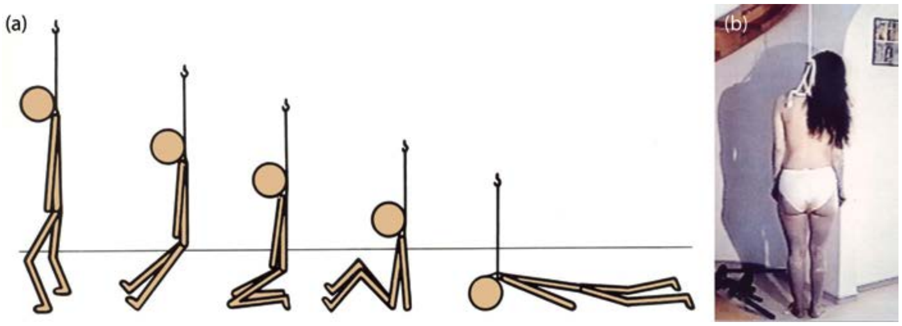


**Figure 1.20** Hanging in different body positions (modified according to Ponsold, 1967).

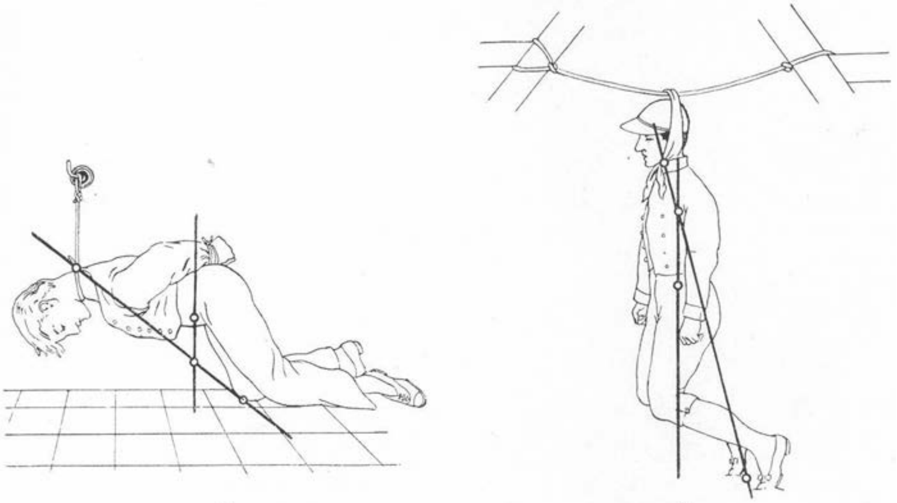


**Figure 1.21** (a), (b) Atypical hanging – calculation of the force on the rope according to Schwarzacher. G = body weight; S = tensile force. (a) G = 65 kg; S = 19 kg. (b) G = 50 kg; S = 42 kg (from Reuter F. *Lehrbuch der Gerichtlichen Medizin*. Berlin, Urban & Schwarzenberg, 1933).

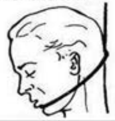
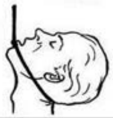
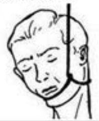
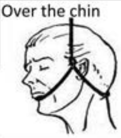
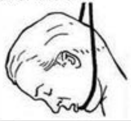
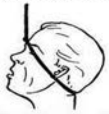
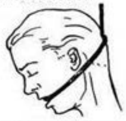
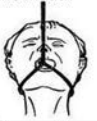
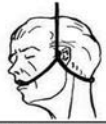
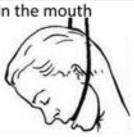
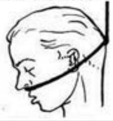
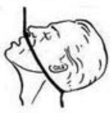
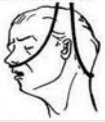
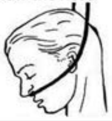
| Position of the rope | Left carotid artery | Right carotid artery | Left vertebral artery | Right vertebral artery | Position of the rope |
|---|---|---|---|---|---|
| Over the chin  | <10 | <10 | 25 (20) | 20 (15) | |
| | 15 (10) | 10 | 15 | 20 (15) | Over the chin  |
| Over the chin  | 20 (10) (15) | 15 (10) | 15 | 15 | |
| | < 10 | 30 (25) | 10 | 10 | Over the chin  |
| Over the chin  | 20 (15) | 15 | At 25 slip off of rope | | |
| | > 35 | > 35 | 10 | 10 (5) | Case Langermann  |
| In the mouth  | < 10 | < 10 | 20 (15) | 15 | |
| | 25 | 38 (35) | < 10 | < 10 | In the mouth  |
| In the mouth  | 15 | < 5 | 35 | <10 | |
| | < 10 | < 10 | 25 (15) (20) | 30 (20) (25) | In the mouth  |
| Below the nose  | 20 | 20 | 20 (15) | 25 (20) | |
| | > 35 | > 35 | 10 | 5 | Below the nose  |
| Below the nose  | > 35 | 15 | > 35 | 10 | |
| | 25 (20) | 30 (20) (25) | > 35 | > 35 | Below the nose  |

**Figure 1.22** Arterial occlusion in supracervical hanging; force in kg necessary to cause a complete arterial compression (after Rauschke, 1957).

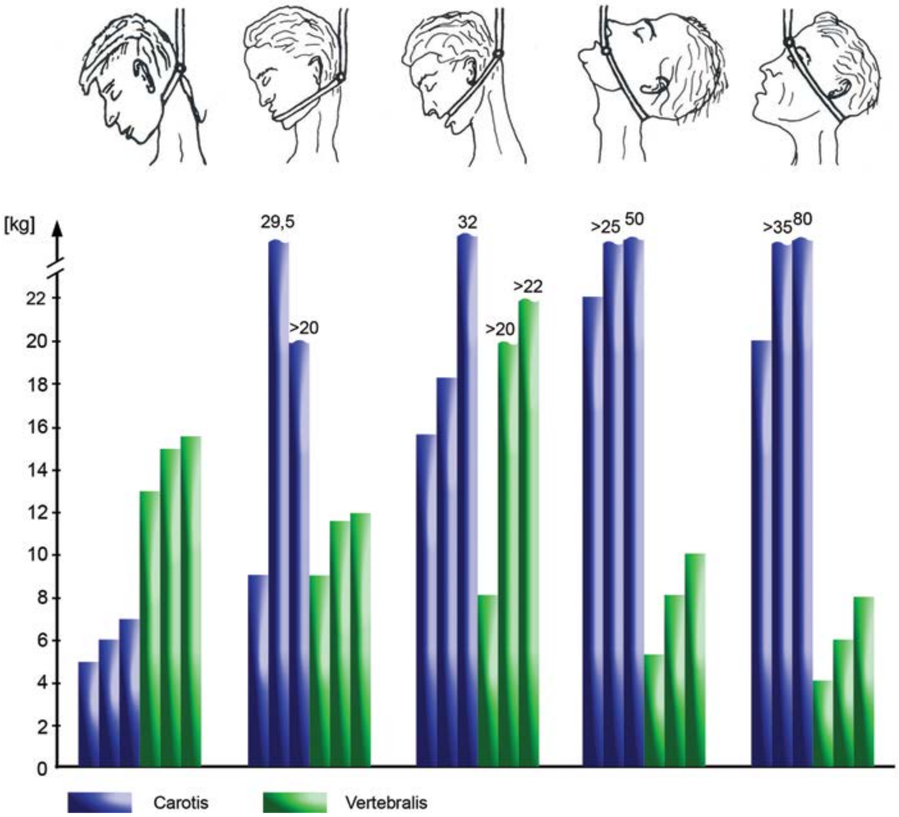
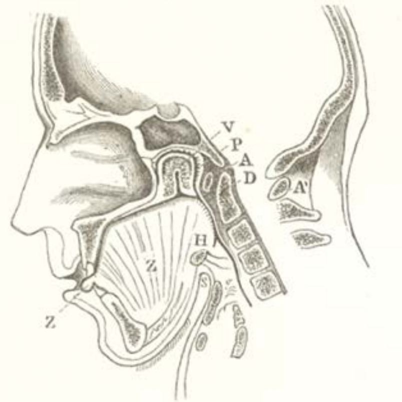


**Figure 1.23** Arterial occlusion depending on the position of the rope; force in kg (Brinkmann B, Püschel K. *Z Rechtsmed* 1981;**86:**175–194; Brinkmann B, Madea B. *Handbuch Gerichtliche Medizin, 2 Bände*. Berlin, Springer, 2004).

Fig. 156.



Sagittaler Durchschnitt durch Kopf und Hals eines gefrorenen Erhängten. (*Ecker* in *Virchows* Arch., 49. Bd., S. 920.) *S* Strangfurche. *H* Zungenbein. *Z* Zunge, zwischen den Zähnen eingeklemmt. *V* weicher Gaumen, in den Nasenrachenraum hinaufgedrängt, *P* hintere Rachenwand. *A* Atlas. *D* Epistropheus.

**Figure 1.24** Sagittal section through the head of a frozen body. Cause of death hanging. The tongue was pressed against the posterior pharynx wall (from Ecker, 1870). S = strangulation mark; H = hyoid bone; Z = tongue; V = pharynx; P = posterior pharynx wall; A = atlas; D = epistropheus.

killing his pregnant lover by ligature strangulation and of subsequently throwing her into water. In 1926 he was sentenced to death. In a later confession he claimed as exonerating evidence the Hering carotid sinus reflex, admitting that he had applied pressure to the neck but without any intention of killing the woman. At court there were intense controversial discussions among the experts and the death penalty was eventually reduced to a lower penalty. Of course, this case was noted and fatal reflectory carotid sinus reflex is not infrequently used as an exclusion in cases of death from pressure to the neck. Meanwhile, recommendations for the investigation of the glomus caroticum in cases of fatal pressure to the neck have been proposed. In court, testimonies concerning instantaneous neurogenic cardiac arrest due to carotid sinus stimulation can normally be ruled out by a thorough evaluation of the autopsy and histological findings.

## Self-experiments on strangulation

The self-experiments on hanging by Professor Nicolae Minivici (1868–1941), who was head of the Institute of Forensic Medicine in Bukarest from 1932 to 1938, are well known. However, a German physiologist, Erich Opitz (1909–1953), also carried out self-experiments. In 1950, the first edition of Albert Ponsold's (1900–1983) *Lehrbuch der*

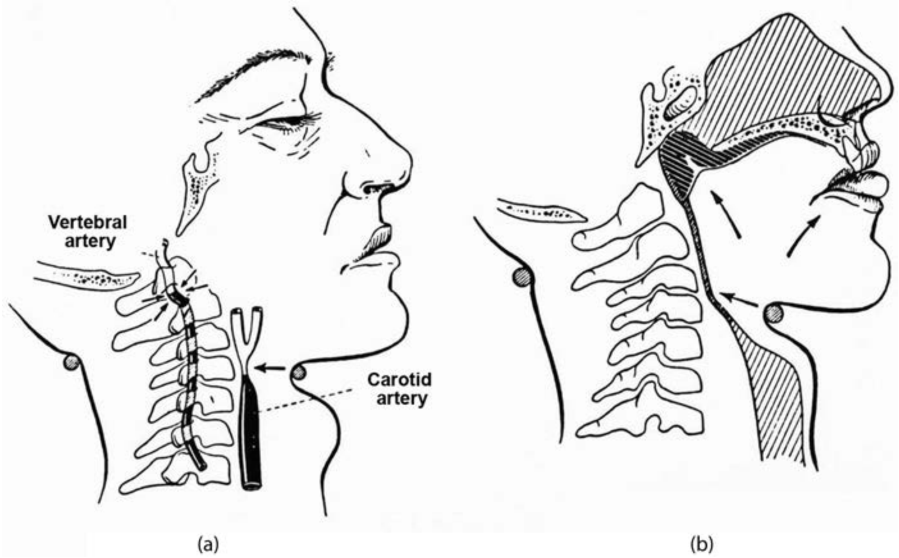
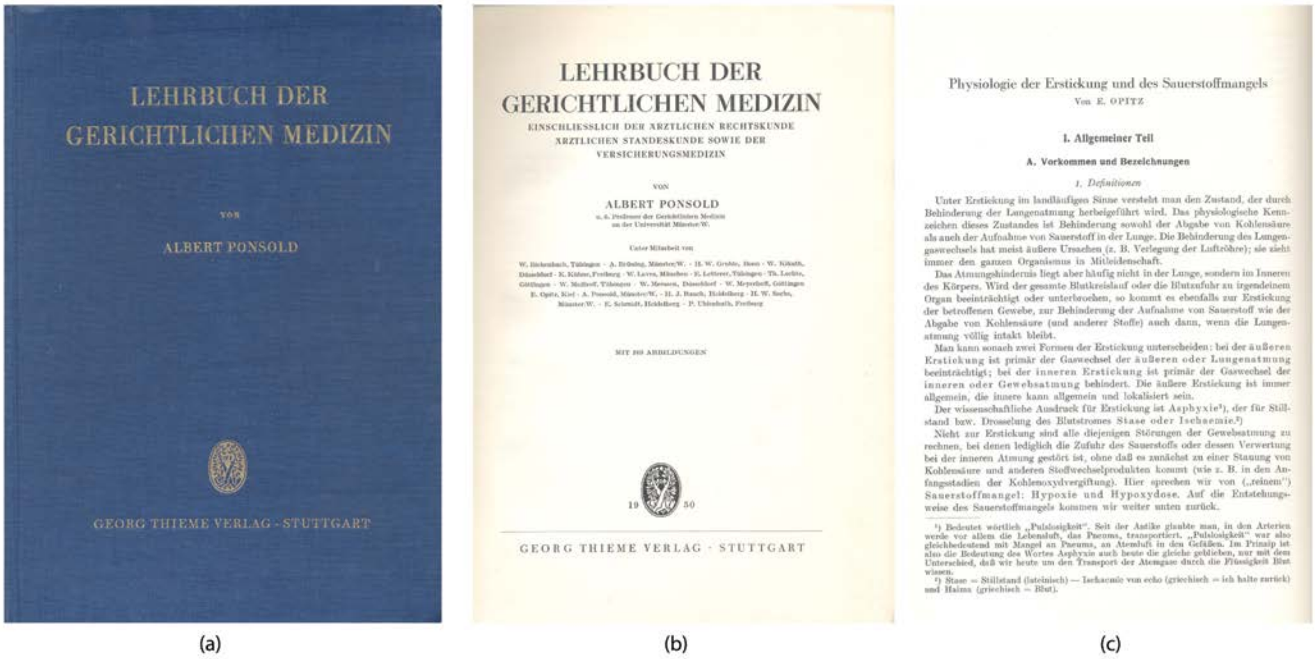


**Figure 1.25** Different mechanisms leading to death by hanging: (a) occlusion of arteries; (b) airway obstruction (from Ponsold A. *Lehrbuch der Gerichtlichen Medizin*. Stuttgart, Thieme, 1950).

*Gerichtlichen Medizin* (Textbook of Forensic Medicine) was published (Figure 1.26a–c). The book became famous for its schematic drawings and these drawings were copied worldwide. In the first edition, the chapter on asphyxiation was written mainly by Opitz. With the pathologist Franz Büchner, he moved from Freiburg to the Berliner Hospital am Friedrichshain. He wrote a dissertation on *'Herzmuskelveränderungen durch Störung der Sauerstoffzufuhr'* ('Alterations of cardiac muscle due to oxygen deficiency'). Together with the physiologist Erich Schütz (1902–1988), he carried out further research on oxygen deficiency and cardiac muscle. In 1941 he received his PhD in Göttingen. The title of the PhD thesis was 'About acute hypoxia'. After World World War II, he worked at the Christian-Albrechts-University in Kiel and, in 1953, was awarded the Chair of Physiology in Göttingen, but he died soon after as the result of an accident.

The Opitz chapter on asphyxia is one of the best ever written in a textbook of forensic medicine. He carried out self-experiments with ligature strangulation of



LEHRBUCH DER
GERICHTLICHEN MEDIZIN
VON
ALBERT PONSOLD
GEORG THIEME VERLAG · STUTTGART

LEHRBUCH DER
GERICHTLICHEN MEDIZIN
EINSCHLIESSLICH DER ÄRZTLICHEN RECHTSKUNDE
ÄRZTLICHEN STANDESKUNDE SOWIE DER
VERSICHERUNGSMEDIZIN
VON
ALBERT PONSOLD
a. o. Professor der Gerichtlichen Medizin
an der Universität Münster/W.
Unter Mitarbeit von
19 50
GEORG THIEME VERLAG · STUTTGART

Physiologie der Erstickung und des Sauerstoffmangels
Von E. OPITZ

I. Allgemeiner Teil

A. Vorkommen und Bezeichnungen

*1. Definitionen*

Unter Erstickung im landläufigen Sinne versteht man den Zustand, der durch Behinderung der Lungenatmung herbeigeführt wird. Das physiologische Kennzeichen dieses Zustandes ist Behinderung sowohl der Abgabe von Kohlensäure als auch der Aufnahme von Sauerstoff in der Lunge. Die Behinderung des Lungengaswechsels hat meist äußere Ursachen (z. B. Verlegung der Luftröhre); sie zieht immer den ganzen Organismus in Mitleidenschaft.

Das Atmungshindernis liegt aber häufig nicht in der Lunge, sondern im Inneren des Körpers. Wird der gesamte Blutkreislauf oder die Blutzufuhr zu irgendeinem Organ beeinträchtigt oder unterbrochen, so kommt es ebenfalls zur Erstickung der betroffenen Gewebe, zur Behinderung der Aufnahme von Sauerstoff wie der Abgabe von Kohlensäure (und anderer Stoffe) auch dann, wenn die Lungenatmung völlig intakt bleibt.

Man kann sonach zwei Formen der Erstickung unterscheiden: bei der äußeren Erstickung ist primär der Gaswechsel der äußeren oder Lungenatmung beeinträchtigt; bei der inneren Erstickung ist primär der Gaswechsel der inneren oder Gewebsatmung behindert. Die äußere Erstickung ist immer allgemein, die innere kann allgemein und lokalisiert sein.

Der wissenschaftliche Ausdruck für Erstickung ist Asphyxie[1]), der für Stillstand bzw. Drosselung des Blutstromes Stase oder Ischaemie.[2])

Nicht zur Erstickung sind alle diejenigen Störungen der Gewebsatmung zu rechnen, bei denen lediglich die Zufuhr des Sauerstoffs oder dessen Verwertung bei der inneren Atmung gestört ist, ohne daß es zunächst zu einer Stauung von Kohlensäure und anderen Stoffwechselprodukten kommt (wie z. B. in den Anfangsstadien der Kohlenoxydvergiftung). Hier sprechen wir von („reinem") Sauerstoffmangel: Hypoxie und Hypoxydose. Auf die Entstehungsweise des Sauerstoffmangels kommen wir weiter unten zurück.

[1]) Bedeutet wörtlich „Pulslosigkeit". Seit der Antike glaubte man, in den Arterien werde vor allem die Lebensluft, das Pneuma, transportiert. „Pulslosigkeit" war also gleichbedeutend mit Mangel an Pneuma, an Atemluft in den Gefäßen. Im Prinzip ist also die Bedeutung des Wortes Asphyxie auch heute die gleiche geblieben, nur mit dem Unterschied, daß wir heute um den Transport der Atemgase durch die Flüssigkeit Blut wissen.

[2]) Stase = Stillstand (lateinisch) — Ischaemie von echo (griechisch = ich halte zurück) und Haima (griechisch = Blut).

(a) (b) (c)

**Figure 1.26** (a) Cover, (b) title page and (c) beginning of the chapter on asphyxiation by Opitz from the famous textbook of forensic medicine of Ponsold A. *Lehrbuch der Gerichtlichen Medizin*. Stuttgart, Thieme, 1950.

the cervical arteries using a blood pressure cuff with a pressure of 250 mm/Hg. This ligature strangulation using a blood pressure cuff showed that, with peracute brain ischaemia in humans, blood pressure is rising and pulse rate is decreasing. After 8 seconds, the person is losing consciousness and suffering convulsions. After reperfusion, the convulsions are still increasing, as is the pulse rate (Figure 1.27).

In addition to self-experiments, observations on death by hanging with a rope during justifications were reported (Figures 1.28 and 1.29). Furthermore, experiments carried out in dogs (Figure 1.30) showed that bradycardia was accompanied by a rise in arterial blood pressure.

The general stages of asphyxiation have long been known (Table 1.14) and have been confirmed by Sauvageau et al. (2009, 2010, 2011), who thoroughly studied agonal sequences in hanging in filmed autoerotic accidents (Table 1.15).

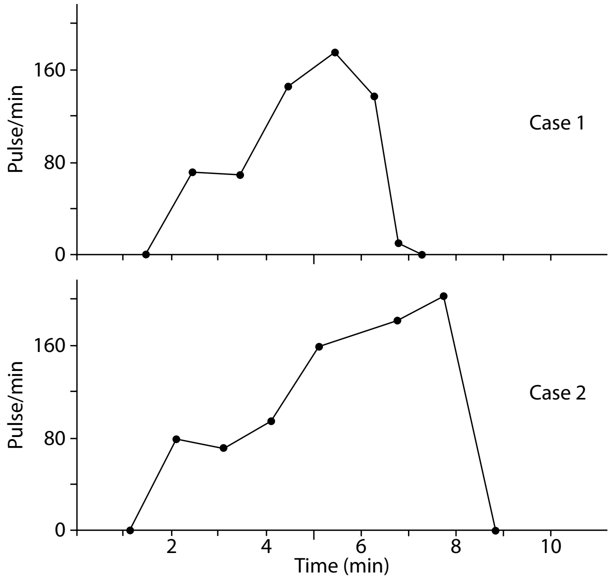


**Figure 1.28** Heart rate profile in hanging. Initial cardiac arrest over 86 and 67 seconds (according to Miloslavich, 1919).

## Autoerotic death

Sauvageau has reviewed the historical context of autoerotic deaths in the forensic literature. One of the first reports on autoerotic death was by Ernst Ziemke (1867–1935) who was head of the Institute of Forensic Medicine in Kiel from 1906 to 1935. In 1925, he reported in the German *Journal of Legal Medicine* 'On accidental strangulation'.

The individuals involved are usually men, originating from all age groups and professional backgrounds, but usually beyond adolescence and middle age. The phenomenon is almost unknown in women. Autoerotic deaths represent fatal accidents ('operating accidents') as a direct consequence of autoerotic activities of single individuals being used for sexual stimulation and masturbation without a sexual partner. In these circumstances, sexual stimulation is provoked and increased by central nervous system stimulation because of a depression of cortical controlling functions (e.g. lack of oxygen, narcotics) or peripheral stimuli, especially to the erogenous zones (e.g. mechanically or by electricity). The sexually stimulating effects of hypoxia in connection with strangulation and

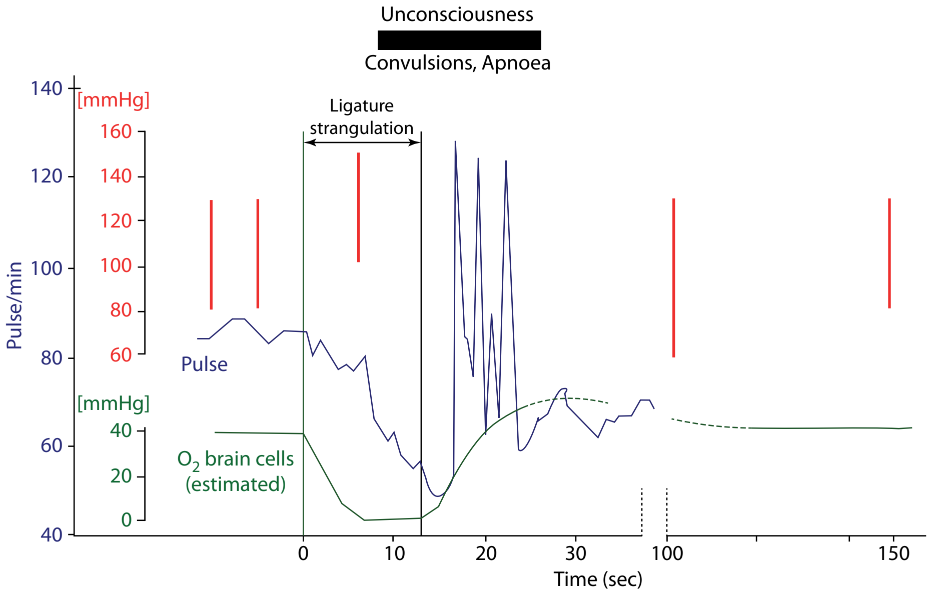


**Figure 1.27** Sudden ligature strangulation of the cervical arteries using a blood pressure cuff with a pressure of 250 mm/Hg. Left *y*-axis: pulse. Red: blood pressure in mm/Hg. *x*-axis: time in seconds. After beginning ligature strangulation, blood pressure is rising and pulse is decreasing. After 8 seconds, there is loss of consciousness. During the loss of consciousness convulsions are experienced. In the recirculation period, the number of convulsions increases, as does the pulse (according to Opitz, 1950).

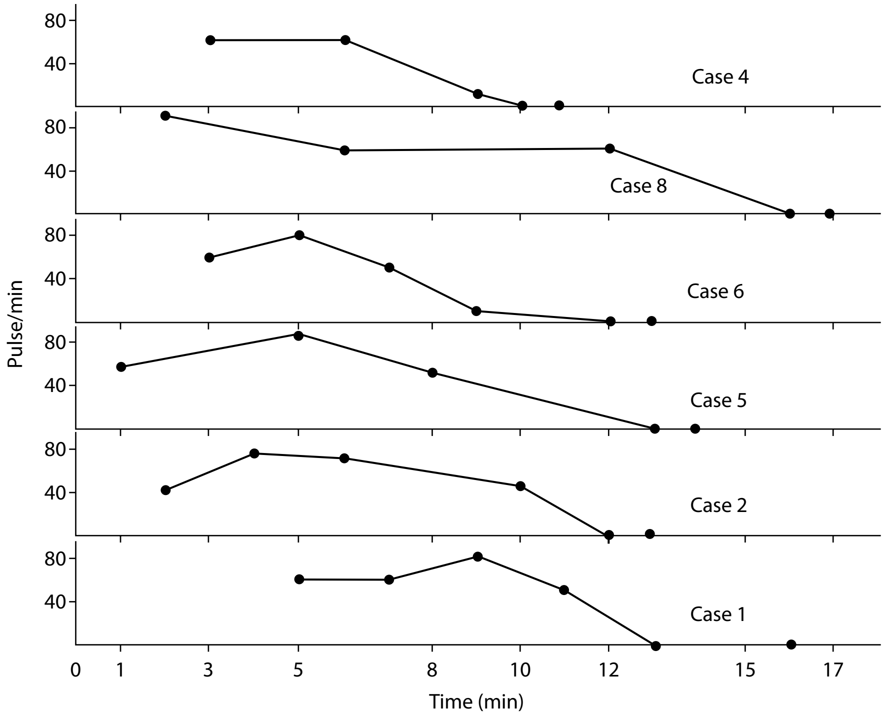


**Figure 1.29** Heart rate profile in hanging (according to Kalle, 1933). There is a 1–5-minute loss of time between the beginning of the hanging and the first measurement of heart rate.

of the use of plastic bags for oro-nasal occlusion have been discussed thoroughly, but the actual mechanism has not yet been clarified. Sometimes, the individuals concerned also aim to achieve a situation of personal fear and pain (masochism). Essentially, erotic fantasies are deliberately induced by partial cerebral ischaemia, mostly achieved by some form of hypoxia or pressure on the neck.

The criteria of autoerotic death proposed by Mant (1960) are shown in Table 1.16.

The best monograph on autoerotic deaths has been written by Sauvageau and Geberth (2013) (Figure 1.31). Drawing on their wide experience, autoerotic death-scene characteristics can be summarized as shown in Tables 1.17 and 1.18.

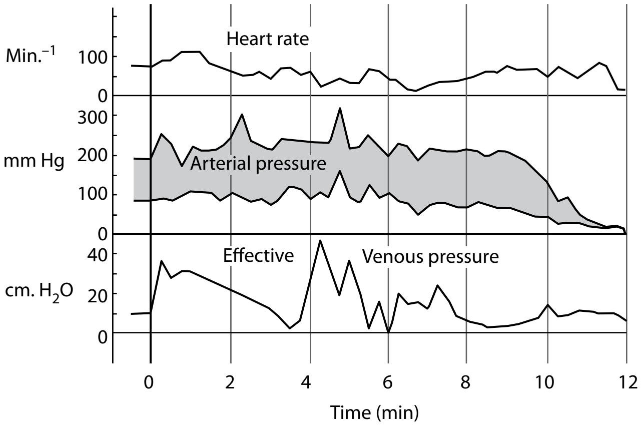


**Figure 1.30** Haemodynamic dysregulation in occlusion of breathing orifices (obstructive asphyxia) (modified from Swann and Brucer, 1949).

**Table 1.14 General stages of asphyxiation (according to Opitz, Ponsold and Berg)**

| Stage | Observations | Approximate duration |
|---|---|---|
| Dyspnoea | 'Air hunger', tachypnoea, deep breathing, use of secondary muscles of respiration<br>Frequently first inspiratory, then expiratory dyspnoea<br>Tachycardia, increase in blood pressure<br>Cyanosis of the face<br>Loss of consciousness | 1 min–1 min 20 s |
| Convulsions | Tonic–clonic convulsions<br>Injuries due to bumps can arise during the convulsions<br>Deep loss of consciousness<br>Mydriasis<br>Further increased, possibly still increasing blood pressure, further tachycardia possible<br>Occasionally release of urine and faeces | 2 min |
| Preterminal apnoea | Temporary apnoea<br>Fall in blood pressure<br>Usually tachycardia | 1 min |
| Terminal agonal respiration | Agonal breaths between which there are long pauses<br>Tensioning of the neck muscles | 1–4 min |
| Apnoea | Irreversible apnoea<br>Sometimes tachycardia, evidently due to hypoxic paralysis of the nucleus of the vagus nerve | Cardiac activity can be maintained for up to 20 min |

**Table 1.15** **Agonal sequence in hanging***

| Event | Average time* |
|---|---|
| Loss of consciousness | 10 ± 3 s |
| Convulsions | 14 ± 3 s |
| Decerebrate rigidity | 19 ± 5 s |
| Start of deep rhythmic abdominal respiratory movements | 19 ± 5 s |
| Decorticate rigidity | 38 ± 15 s |
| Loss of muscle tone | 1 min 17 s ± 15 s |
| End of deep rhythmic abdominal respiratory movements | 1 min 51 s ± 30 s |
| Last muscle movement | 4 min 12 s ± 2 min 29 s |

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**Table 1.16** **Proposed criteria for the diagnosis of autoerotic death**

| | |
|---|---|
| 1 | Evidence of asphyxia produced by strangulation, by either ligature or hanging, in which the position of the body, or the pressure of the protective measures, such as padding about the neck, indicated that death was not obviously intended |
| 2 | Evidence of sexual activity usually in the form of masturbation or perversion, especially transvestism |
| 3 | No well-defined evidence for suicide or minimal evidence of suicidal ideation or behaviour |
| 4 | Evidence or repetitive episodes |

*Source:* Mant, 1960.

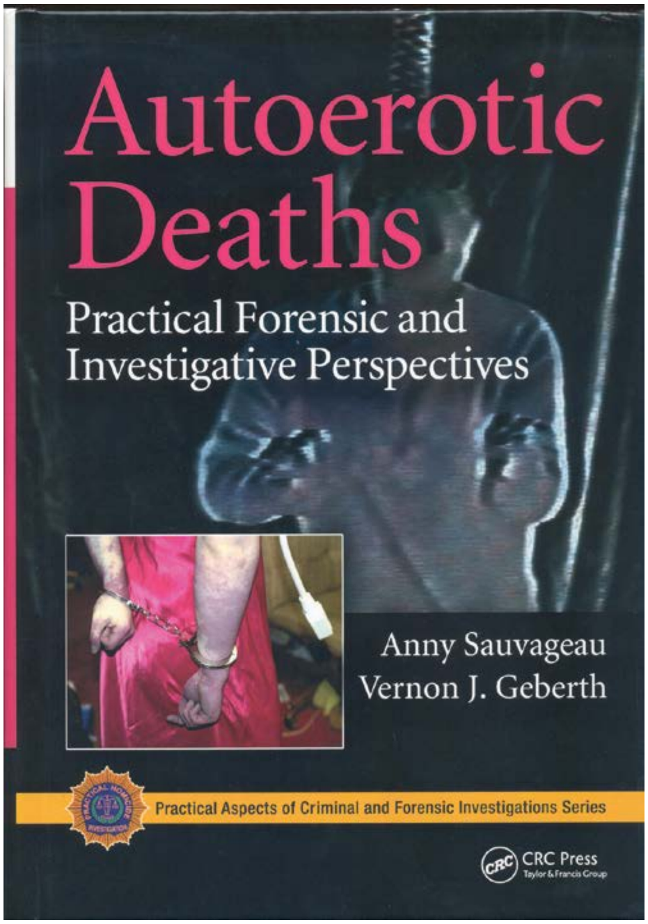

**Figure 1.31** Cover of Sauvageau and Geberth's 2013 monograph on autoerotic deaths.

## Vital reactions

Vital reactions have been a main research topic of continental forensic medicine for a considerable time, and many famous forensic pathologists have devoted their research and contributed important papers to this field. The area of research ranges from macroscopically visible organ reactions over tissue alterations (enzyme histochemistry, plus immunohistochemistry with a wide range of enzymes and other analytes) to biochemical responses to injury (e.g. catecholamines in various body fluids, thyroglobulin, histamine content of the strangulation mark, positive aquaporin-3 staining of the strangulation mark).

Vital reactions are, of course, of great importance in cases of fatal strangulation.

Numerous continental European textbooks include large sections on vital reactions. According to Bernhard Knight (1996), the 'vital reaction is a dubiously valid phenomenon, mainly because death and dying is a process, not an event'.

The question of whether an injury was inflicted during life is one of the most important subjects in forensic medicine, especially in asphyxial deaths. Any effects in, at or by the body following trauma are named 'vital reaction', allowing the conclusion that trauma was inflicted during life. The term 'vital reaction' can be subdivided into 'vital reactions', 'vital processes' and 'vital signs' (Orsos, 1935; Madea et al., 2014) (Table 1.19).

- *Vital reactions* are local reactions of tissues at the sites of damage.
- *Vital processes*, conversely, are reactions involving the whole organism and not only local cells and tissues. The reaction of the organism requires functioning of the nervous, respiratory and cardiovascular systems.
- *Vital signs* are findings which allow the conclusion of the vital origin of a trauma (blood stain pattern in the form of arterial blood spray, aspiration or swallowing of blood). Local vital reactions at the site of trauma must be differentiated from systemic vital reactions of the great organ systems (especially circulatory and respiratory systems; Table 1.20). In fatal injuries, the survival time after trauma is often short, shorter than the manifestation time of local vital reactions at the site of injury. The manifestation of a local vital reaction at the site of injury (e.g. immigration of polymorphnuclear leucocytes) requires some time. Systemic vital reactions, especially of the circulatory and respiratory systems, develop very fast and are

**Table 1.17 Autoerotic death-scene characteristics***

| **Autoerotic death-scene characteristics: Checklist for the forensic expert** |
|---|
| Know that the death scene is the most important part in establishing a death as autoerotic. |
| Never interpret the death scene in isolation; also consider the external examination or autopsy findings and the history. |
| Know the 15 scene characteristics that can be found in autoerotic deaths: nudity, exposure of the genitals, cross-dressing, evidence of masturbatory activity, foreign body insertion in the anus, lubricants, pornography, mirror, video recording, covering of the face (e.g. mask, duct tape), bondage of the genitals, other bondage, other masochistic behaviour, protective padding in hanging, and evidence of repetitive behaviour. |
| Be aware that exposed genitals or nudity is not mandatory for a diagnosis of autoerotic accident. |
| Be aware that evidence of masturbatory activities is not mandatory for a diagnosis of autoerotic accident; on the contrary, it is a rare feature. |
| Be aware that the mere presence of semen on the penis or thighs is not necessarily a sign of masturbation with ejaculation. |
| Do not overinterpret the presence of stored pornographical material. |
| Pay particular attention to pornographic material of a particular nature: material on autoerotic death, bondage or masochism. |
| Never rule a death as autoerotic if there are clear indications at the scene of a suicidal or homicidal manner of death. |
| Know that the most common death-scene features in autoerotic deaths are exposure of genitals (66%), pornography (42%), nudity (41%), cross-dressing (39%) and bondage (37%). |
| Be aware that protective padding is present in only one in five autoerotic hangings. |
| Know that mirror, video recording and evidence of masturbation activity are overall uncommon scene features. |
| Expect on average only three scene features. |
| Know that the most common combination of scene features is exposed genitals, nudity and pornography. |
| Be aware that the presence of only one classic scene feature is sufficient for ruling a death as autoerotic if that is the conclusion that seems the most appropriate after completing a full death investigation, including body examination and history review. |
| For victims younger than 20 years of age, expect on average only two scene features. |

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**Table 1.18 Autoerotic death by smothering by plastic bags over the head and by suffocation using chemical substances***

| **Autoerotic death by smothering by plastic bags over the head: Checklist for the forensic expert** |
|---|
| In a scene of suffocation by plastic bag, take particular care to describe and document the type of plastic bag; the position of the bag in relation to the head, mouth, and nose; if the plastic bag is secured at the neck; if a ligature is present to enclose the bag; the type of ligature, its loops and knots; the knots on the plastic bag. |
| Do not remove the plastic bag from the head before proper photographic documentation if the victim is obviously dead. |
| Be aware that, without proper scene information, the diagnosis of smothering by a plastic bag over the head is likely to be missed. |
| Know that the autopsy findings are nonspecific. |
| **Autoerotic death by suffocation using chemical substances: Checklist for the forensic expert** |
| Know that the most commonly used chemical compounds in the context of autoerotic practice are hydrocarbons, anaesthetic compounds and other chemical inhalants. |
| Pay particular attention to odours when entering a scene. |
| At the scene, describe the presence of gas tank, mask, plastic bag or item of clothing on the face or head of the victim or beside the body. |
| Know that plastic bags possibly containing chemicals can be submitted for toxicology (bags should be put in an airtight can or glass jar [no plastic container should be used] and refrigerated). |
| Be aware that pressurized cans found at the scene should be seized for toxicological analysis. |
| Be aware that some suffocation gases (e.g. helium) cannot be tested, and that scene investigation is extremely important to establish the cause of death. |

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**Table 1.19 Definitions by Orsos, 1935 (according to Madea et al., 2014)**

| Term | Definition |
|---|---|
| Vital reactions | Local reactions of tissue at the site of damage |
| Vital processes | Reaction of the whole organism, not only cells and tissues are damaged; reaction of the organism requires function of the nervous, respiratory and cardiovascular systems |
| Vital signs | Findings which allow a conclusion of vital origin (blood stain pattern in form of arterial blood spray; aspiration or swallowing of blood) |

often the main proof for the vitality of injuries. Several influencing factors on the time course of local vital reactions are known (Table 1.21). In cases of fatal strangulation, the systemic vital reactions of the circulatory and respiratory systema are of particular importance.

## Circulation

### *Haemorrhages*

Internal or external haemorrhages are seen in a variety of traumatic deaths. They are mainly due to lesions of arteries, veins or capillaries. Diapedetic haemorrhages or haemorrhages due to coagulation disorders are comparatively rare. Haemorrhages due to lesions of arteries or veins are normally seen at the site of trauma (Table 1.22).

Capillary haemorrhages, like petechial haemorrhages or ecchymoses, can also be seen distant from the site of injury. Petechial haemorrhages are due to an intravascular rise of

**Table 1.20 Vital reactions of different organ systems (systemic vital reactions) (according to Madea et al., 2014)**

| Organ system | Systemic vital reactions |
|---|---|
| Circulatory system | Exsanguination/petechial haemorrhages/ embolisms: air, fat, tissue, bone marrow, foreign bodies (e.g. bullet fragments) |
| Respiratory system | Aspiration (chyme, foreign bodies, blood, brain tissue, soot, water or other fluids)<br>Alveolar–capillary diffusion (gas; detection of gas in the circulation)<br>Emphysema acuta (e.g. emphysema aquosum seen in drowning)<br>Emphysema of the skin |
| Gastrointestinal tract | Vomiting/swallowing<br>Peristaltic transport of stomach contents<br>Absorption/resorption of detectable substances<br>Gastric mucosal erosions (e.g. Wischnewsky ulcers seen in hypothermia) |
| Endocrine system | Agonochemical stress reaction with increase of catecholamine levels |
| Nervous system | Crow's feet-like pattern/secretion of saliva and mucus |

**Table 1.21 Factors possibly influencing the time course of local vital reactions after tissue damage**

| Factors | Influence |
|---|---|
| Local | Manner and intensity of trauma<br>Severity and extension of the alteration<br>Type of the affected tissue<br>Temperature<br>Blood circulation (e. g. reduction of peripheral circulation during shock) |
| General | Hereditary factors, age, gender<br>Nutritional status<br>Additional diseases<br>Endocrine influences<br>Vegetative influences<br>Metabolic disorders |
| Exogenous | Pharmaceuticals<br>Vital hypothermia/hyperthermia |

**Table 1.22 Vital reactions – haemorrhages (according to Madea et al., 2014)**

| Phenomenon | Mechanism | Detection | Postmortem origin |
|---|---|---|---|
| Arterial/ venous haemorrhage | Lesions of vessels, blood extravasation corresponding to the pressure drop intravasal/extravasal | Macroscopic | Yes |
| Capillary haemorrhage | Intracapillary pressure rise caused by congestion with increase of the transvascular pressure gradient | Macroscopic | Yes |

*Note:* The degree of haemorrhage depends on the size of the injured vessel, the blood pressure and the resistance to the blood that is streaming.

pressure with an increasing transvascular pressure gradient from the inside to the outside of the vessel. The higher the intravascular pressure, the shorter the manifestation time until petechial haemorrhages develop. Petechial haemorrhages are of great medicolegal significance regarding the following questions:

- Assessing the vitality of the injury or injuries (e.g. pressure to the neck).
- Deciding whether the compression of the neck was life-threatening.
- Ascertaining the priority of injuries (e.g. pressure to the neck first and then stabbing, or vice versa).
- Distinguishing the type of pressure to the neck between reflex death and manual or ligature strangulation.

Petechial haemorrhages may be found in skin and mucosa (Figure 1.32, Table 1.23). One of the first forensic scientists to study subconjunctival petechial haemorrhages intensively was Otto Prokop (Table 1.24) (Prokop and Wabnitz, 1970). The frequency of petechial haemorrhages in asphyxia death is shown in Table 1.25.

A detailed literature review in 1990 by Geserick and Kämpfe and an evaluation of autopsies in 500 individuals in whom asphyxia was the cause of death revealed the following information (see also Table 1.26):

1. Petechial haemorrhages in the region of the head and neck are not asphyxial haemorrhages because they are the result not of oxygen deficiency but of an increase in intravascular pressure.

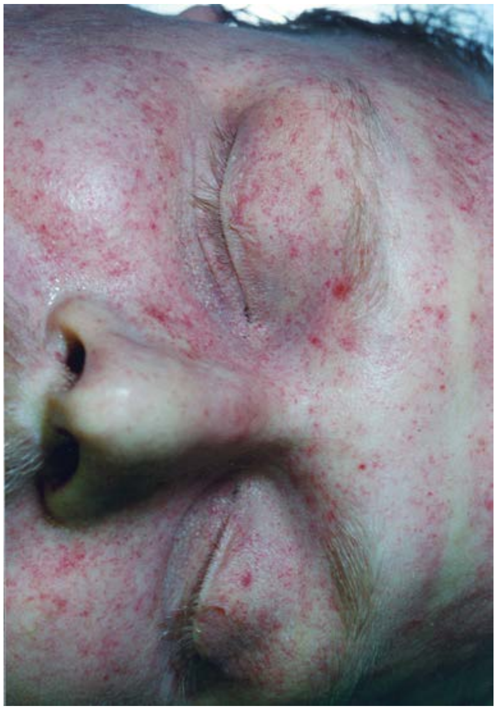

**Figure 1.32** Petechiae of the face.

**Table 1.23 Localization of petechial haemorrhages (according to Geserick and Kämpfe, 1990)**

| Examination | Haemorrhages found |
|---|---|
| External | Mucosa:<br>• Eye<br>• Nose<br>• Lips<br>• Mouth<br>• Tongue<br>• Pharynx<br>• Tonsils<br>• Tympanic membrane<br>Skin:<br>• Eyelids<br>• Forehead<br>• Cheeks<br>• External auditory canal<br>• Behind ears<br>• Neck |
| Internal (autopsy) | Tonsils<br>Tongue<br>Thyreoidea<br>Salivary glands<br>Epiglottis<br>Paranasal sinuses<br>Galea<br>Temporal muscle<br>Retina |

**Table 1.24 Frequency of petechial hemorrhages without asphyxial death**

| Cause | Frequency (%) |
|---|---|
| In fatal cases: | |
| • Electrocution | 12.5 |
| • Central death | 7.6 |
| • Haemorrhagic diathesis | 6.6 |
| • Sudden cardiac death | 4.3 |
| • Sepsis | 3.5 |
| • Intoxication by sedatives | 2.9 |
| • CO intoxication | 2.3 |
| In living cases: | |
| • Delivery | 24.5 |
| • Cough | 3.0 |
| • Vomiting | 0.8 |

*Source:* Prokop O, Wabnitz R. *Z Rechtsmed* 1970;**67:**249–257.

2. Among these haemorrhages, subconjunctival petechiae play the most important role in the practice of forensic medicine.
3. Quantity, intensity and location must be considered when estimating petechial haemorrhages (Table 1.27).
4. Congestive petechiae are not inevitable in cases of obstructive asphyxia. They are often missing in cases of aspiration, typical hanging, and suffocation without neck compression (e.g. by obstruction of the airway). They are nearly always present in cases of neck compression and traumatic asphyxia.

**Table 1.25 Frequency of petechial haemorrhages in asphyxial deaths (according to Geserick and Kämpfe, 1990)**

| Cause of death | Frequency (%) | Author |
|---|---|---|
| Throttling, ligature strangulation | 100 | Bschor, 1969 |
| | 100 | Prokop and Wabnitz, 1970 |
| | 93.4[*] | Haarhoff, 1971 |
| | 100[**] | Wolff, 1984 |
| Chest compression without injury of inner organs | 100 | Prokop and Wabnitz, 1970 |
| Chest compression with injury of inner organs | 25 | Prokop and Wabnitz, 1970 |
| Hanging | 66 | Bschor, 1969 |
| | 64.9 | Lünenbürger, 1954 |
| | 50 | Fritz and Dotzauer (cited from Jacob, 1957) |
| | 47.6 | Laiho et al., 1968 |
| | 38.6 | Schmidt (cited from Jacob, 1957) |
| | 34 | Prokop and Wabnitz, 1970 |
| | 20–30 | Reuter, 1922 |
| | 9 | Martineck (cited from Jacob, 1957) |
| Drowning | 13 | Bschor, 1969 |
| | 3.8 | Prokop and Wabnitz, 1970 |
| Asphyxiation or bolus death | 12 | Bschor, 1969 |
| | 10 | Prokop and Wabnitz, 1970 |

[*] Negative: 2 newborns, 2 infants.

[**] Only in fatal cases or loss of consciousness.

'False-negative' findings can arise from:

- Prolonged survival time.
- Prolonged postmortem period (putrefaction).
- Competing loss of blood, exsanguination.
- Atypical strangulation (by hand or by ligature).

1. Petechial haemorrhages are not specific for obstructive asphyxia. 'False-positive' findings arise as a result of:
    - Natural process (physiological, e.g. during delivery, vomiting, coughing spells, in neonates; pathological, e.g. haemorrhagic diatheses).
    - Unnatural impact (e.g. electric current, poisonings).
    - Postmortem development (hypostasis, exposure to heat).
2. Petechial haemorrhages are important in forensic medicine when assessing:
    - The vital reaction of the organism.
    - The danger to life or the duration and intensity of a compressing force. The question as to the period necessary to produce petechial haemorrhages is still at issue. Experiments to produce congestive pressure in the neck–head region can only be done with animals and require about 3 minutes in the case of experimental suction. Where high

**Table 1.26 Petechial haemorrhages of the head (Institute of Forensic Medicine, Humboldt University, Berlin: 500 cases of fatal asphyxia) (according to Geserick and Kämpfe, 1990)**

| Cause of death | *N* | Positive *n* | Percentage positive (%) | Degree |
|---|---|---|---|---|
| Ligature strangulation | 14 | 14 | 100.0 | 0.9 |
| Chest compression | 14* | 13 | 92.9 | 0.86 |
| Throttling | 9** | 7 | 77.8 | 0.67 |
| Hanging | 302 | 132 | 43.7 | 0.25 |
| Trunk compression | 14 | 5 | 35.7 | 0.31 |
| Suffocation | 17 | 3 | 17.6 | 0.12 |
| Bolus death | 19 | 3 | 15.8 | 0.07 |
| Drowning | 82 | 7 | 8.5 | 0.05 |
| Aspiration | 29 | 2 | 6.9 | 0.03 |
| **Total** | **500** | **186** | **37.2** | **0.24** |

* 1 negative case: Polytrauma with haematothorax.
** 1 negative case = putrefaction *and* 1 negative case = neck compression of the left side.
*Scoring:*
No petechial haemorrhages = 0 points
Slight petechial haemorrhages = 1 point
Middle petechial haemorrhages = 2 points
Strong petechial haemorrhages = 3 points

**Table 1.27 Petechial haemorrhages to the head according to localization and cause of death (according to Geserick and Kämpfe, 1990)**

| Cause of death | *n* | C | C and L and F | C and F | C and L | L | F |
|---|---|---|---|---|---|---|---|
| Ligature strangulation | 14 | 3 | 9 | 1 | 1 | – | – |
| Chest compression | 13 | 1 | 8 | 2 | 1 | 1 | – |
| Throttling | 7 | – | 4 | 3 | – | – | – |
| Hanging | 132 | 64 | 29 | 16 | 19 | 4 | – |
| Trunk compression | 5 | 1 | 3 | – | – | 1 | – |
| Suffocation | 3 | 1 | 1 | – | – | – | 1 |
| Bolus death | 3 | 2 | – | 1 | – | – | – |
| Drowning | 7 | 6 | – | 1 | – | – | – |
| Aspiration | 2 | 1 | – | 1 | – | – | – |
| **Total (%)** | **186 (100.0)** | **79 (42.5)** | **54 (29.0)** | **25 (13.5)** | **21 (11.3)** | **6 (3.2)** | **1 (0.5)** |

*Note:* C = conjunctivae; L = lips and mouth mucosa; F = face skin.

intravascular peaks of pressure are involved, such as coughing, vomiting, or crushing pains, much less time is required (10–20 seconds).

- ‘Reflex deaths’.
- The priority (sequence) of different impacts.

Using these criteria, a clear and absolute evidential value cannot be attributed to petechial haemorrhages (see points 4 and 5). To use petechial haemorrhages in diagnostics requires an assessment of the entire constellation of findings (inclusive of clinical and criminalistics findings). Provided that they are carefully examined and critically assessed, congestive haemorrhages are still the principal component of the findings and can possibly direct the medicolegal expert in the diagnosis of obstructive asphyxia. Despite all efforts to be objective, the expert’s own experience will continue to play an important role in that process.

In survived strangulation, the intensity and frequency of petechial haemorrhages is greater in the group with loss of consciousness (Table 1.28) than without loss of consciousness (Table 1.29).

In strangulation cases, the priority of injuries is, of course, of great importance.

Fifty-three cases of homicide inclusive of strangulation by hand or by ligature selected from autopsies performed between 1979 and 1988 were analysed by Lignitz and Henn (2007). The deaths, reflecting known strangulation stigmata, included 6 cases of manual strangulation, 6 cases of strangulation by ligature, and 17 cases with a combination of strangulation and other violence to the neck. Finally, 24 cases of death by obstructive asphyxia and other forms of traumatic violence were considered: 12 were combined with blunt traumatic violence, 10 with traumatic violence using a sharp instrument, 1 with burns and 1 with drowning.

**Table 1.28 Intensity and frequency of petechial haemorrhages in cases of survived obstructive asphyxia with loss of consciousness, female $n = 32$ (1980–1988)**

| | Eyelid (%) | Conjunctiva (%) | Skin face (%) | Oral mucosa (%) | All regions (%) |
|---|---|---|---|---|---|
| Number | 37 | 41 | 44 | 53 | 22 |
| Single | 19 | 31 | 12 | 32 | 78 |
| Several | 25 | 9 | 16 | 6 | |
| Massive | 19 | 19 | 28 | 9 | |

*Source:* Strauch H, Lignitz E, Geserick G. In: Brinkmann B, Püschel K (eds). *Ersticken. Fortschritte in der Beweisführung*. Berlin, Springer, 1990, pp 248–255.

**Table 1.29 Intensity and frequency of petechial haemorrhages in cases of survived obstructive asphyxia without loss of consciousness, female $n = 42$ (1980–1988)**

| | Eyelid (%) | Conjunctiva (%) | Skin face (%) | Oral mucosa (%) | All regions (%) |
|---|---|---|---|---|---|
| Number | 69 | 55 | 72 | 73 | 45 |
| Single | 12 | 26 | 2 | 17 | 55 |
| Several | 12 | 12 | 14 | 5 | |
| Massive | 7 | 7 | 7 | 5 | |

*Source:* Strauch H, Lignitz E, Geserick G. In: Brinkmann B, Püschel K (eds). *Ersticken. Fortschritte in der Beweisführung*. Berlin, Springer, 1990, pp 248–255.

The analysis of those 24 cases of compression of the neck in combination with other types of injuries (using sharp and blunt objects) allows the following conclusions:

- Vital strangulation is the most common form of obstructive asphyxia. Being the most sensitive reaction, petechial haemorrhages of the conjunctivae linked with the local lesions are of greatest significance (Prokop and Wabnitz, 1970; Haarhoff, 1971).
- Intensive conjunctival petechiae resulting from obstructive asphyxia in the presence of bleeding wounds ('blood escape opening') or internal bleeding injuries (post-haemorrhagic anaemia) prove violent force transmitted to the neck prior to other kinds of traumatic violence. In cases of bleeding wounds of the head and neck, petechiae are inhibited due to a lack of increase in intracapillary pressures.
- Strangulations (see Brinkmann et al., 1981) are conceivable without conjunctival petechiae if a strangulation mechanism according to the local stigmata does not show any indication of congestion as a vital sign and if the absence of it can be explained by previous bleedings (neck and head wounds). This does not allow the absolute conclusion that strangulation took place post mortem. It can even be the actual cause of death.
- Manual strangulation is often the first attack. Suitable clothing (neckerchiefs, scarfs, garments) offer the possibility of strangulation by ligature as the first aggressive action.
- The presence of petechiae in cases of free suspension evokes suspicion of strangulation by ligature followed by hanging.
- Postmortem strangulation by ligature could expel blood from the vessels by pushing the compressed neck soft tissue upwards and thus simulating the vitality of petechial haemorrhages (Schröder and Saternus, 1983).
- Neck compression with few or no petechial haemorrhages requires a well-considered, extensive and cautious interpretation in the medicolegal report.

However, exsanguination with anaemia of the body does not occur any more.

Of special importance are haemorrhages of the tongue (Figure 1.33, Tables 1.30 and 1.31).

Exsanguination of a body with anaemia of the internal organs is always a vital reaction. Little extent and intensity of postmortem lividity, anaemia of the body, the skin and mucous membranes together with subendocardial bleedings are typical findings of fatal haemorrhages. Further typical vital haemorrhages are so-called 'Simon's haemorrhages', to be found over the lumbar spine in cases of typical hanging (Figure 1.34), haemorrhages of the tongue in long-lasting manual or ligature strangulation, congestion, cyanosis and petechial haemorrhages above the ligature in atypical hanging (Figure 1.35), and

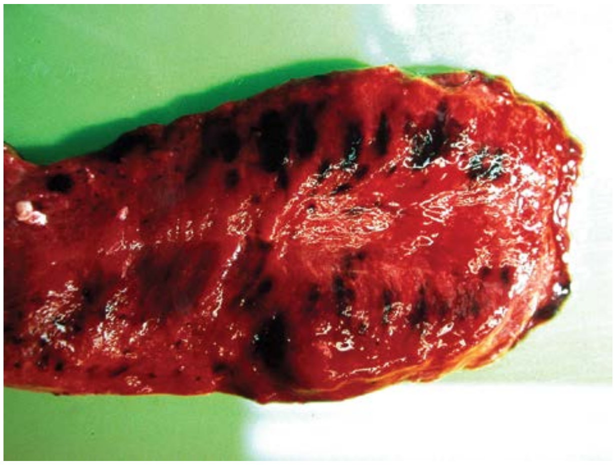

**Figure 1.33** Haemorrhages of the tongue in a case of long-lasting ligature strangulation.

**Table 1.30 Frequency of tongue haemorrhages from the literature**

| Type of strangulation | Number of cases | Author | Number respectively percentage of tongue haemorrhages (%) |
|---|---|---|---|
| Man. str., retrospect. A | 30 | Pollak, Misslewitz, 1985 | 37 |
| Homicidal lig. str., retrospect. A | 19 | Pollak, Misslewitz, 1985 | 10 |
| Homicidal lig. str., prospect. A | 29 | Pollak, Misslewitz, 1985 | 25 |
| Homicidal combined str., retrospect. A | 11 | Suyama et al., 1982 | 18 |
| Homicidal str., prospect. A | 10 | Pollak, Misslewitz, 1985 | 90 |
| Homicidal str., prospect. A | 30 | Simonsen, 1989 | 1 |
| Suicidal lig. str., retrospect. A | 7 | Koops et al., 1982 | 6 |
| Suicidal lig. str., retrospect. A | 12 | Madea, Brinkmann, 1985 | 6 |
| Hanging, prospect. A | 124 | Laiho et al., 1968 | 14 |
| Hanging, prospect. A | 6 | Suyama et al., 1982 | 3 |
| Hanging, prospect. A | 29 | Pollak, Misslewitz, 1985 | 10 |
| Hanging, prospect. A | 80 | Simonsen, 1989 | 0 |

*Source:* Reprinted from Bockholdt B, Maxeiner H. *Forensic Sci Int* 2002;**126:**214–220, with permission from Elsevier.
*Abbreviations:* str. = strangulation; man. = manual; lig. = ligature; retrospect. A/prospect. A = retrospective/prospective analysis.

**Table 1.31 Frequency of haemorrhages and/or bitemarks in the tongue in suicidal and homicidal strangulations**

| | Completely unremarkable | Bitemarks of the surface | Small haemorrhages | Significant haemorrhages | Massive haemorrhages |
|---|---|---|---|---|---|
| Manual str. (*n* = 57) | 11(19) | 16(28) | 19(33) | 9(16) | 2(4) |
| Ligature str. (*n* = 50) | 21(42) | 8(16) | 9(18) | 8(16) | 4(8) |
| Combined str. (*n* = 42) | 13(31) | 6(14) | 8(19) | 13(31) | 2(5) |
| Other types of neck compression (*n* = 29) | 6(21) | 4(14) | 12(41) | 6(21) | 1(3) |
| All homicides (*n* = 178) | 51(29) | 34(19) | 48(27) | 36(20) | 9(5) |
| All suicides (*n* = 275) | 153(92) | 3(1) | 8(3) | 8(3) | 3(1) |
| Suicidal hanging (*n* = 255) | 243(95) | 3(1) | 4(2) | 3(1) | 2(1) |
| Suicidal str. by ligature (*n* = 20) | 10(50) | 0 | 4(20) | 5(25) | 1(5) |

*Source:* Reprinted from Bockholdt B, Maxeiner H. *Forensic Sci Int* 2002;**126:**214–220, with permission from Elsevier.
*Abbreviation:* str. = strangulation.

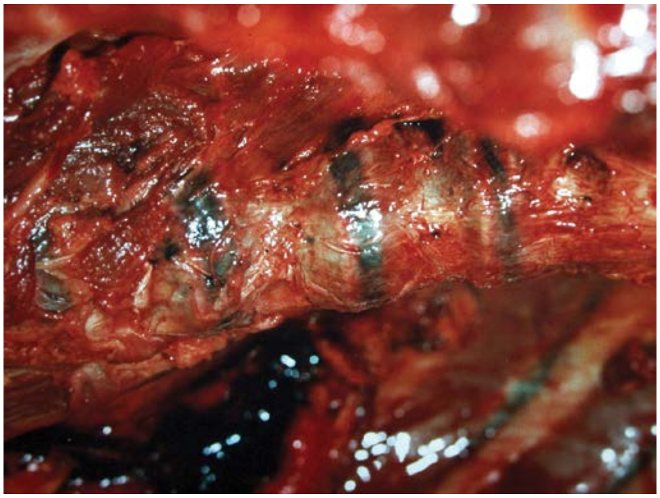

**Figure 1.34** Haemorrhages of the intervertebral discs (Simon's haemorrhages) in a case of typical hanging.

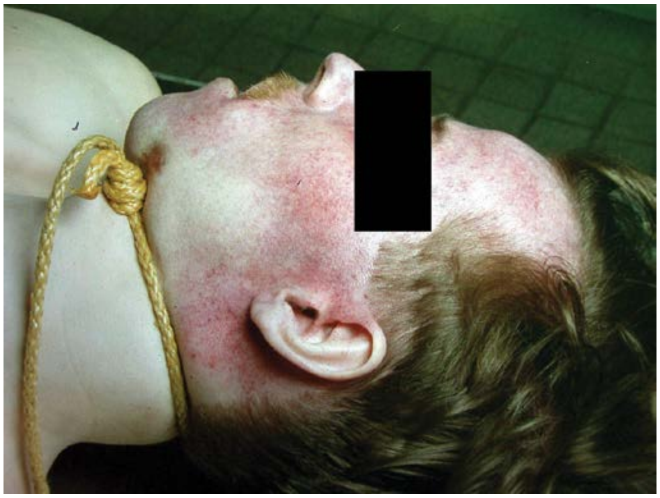

**Figure 1.35** Cyanosis and swelling of the face, massive congestion and petechial haemorrhages in a typical hanging.

intramedullary haemorrhage of the cervical spinal cord due to contusion of the neural tissue.

### *Simon's bleedings as a vital sign of hanging*

In 1968, the German forensic pathologist Axel Simon described for the first time intervertebral haemorrhages as a vital sign of hanging. They were seen mainly in cases of free suspension, in both sexes, in all investigated age groups, but predominantly in younger ages. They were also observed at the dorsal intervertebral discs. Since then, Simon's bleedings have been verified and endorsed by other authors. They occur in 40–50 per cent of hanging cases, most frequently in the lumbar spine, younger age groups and in cases of free suspension. They can, however, also be seen in cases of hanging where there is contact with the ground. Further research has revealed that haemorrhages are not unique to hanging but may occur as a result of other traumatic elongation or overextension of the spinal column (e.g. in the course of traffic accidents). In cases of decomposition of the body, 'false positive' findings are relatively common. The source of the bleedings is capillaries of collaterals of the ramus spinalis of the arteria vena lumbalis.

Since external visible vital signs in cases of hanging may be scarce or completely absent, there is still a need for autopsies since Simon's bleedings can only be seen in an autopsy.

According to Geserick et al. (2012), Simon's bleedings as a vital sign in death by hanging can be found in 26–90 per cent of cases (Table 1.32).

Hejna and Rejtarova (2010) carried out a prospective analysis of 2226 autopsies and found Simon's bleedings in 65 out of 178 cases of hanging and in 17 cases in a group of 350 controls with various causes of death. Based on their study, they have drawn the following conclusions:

- Simon's sign can be evaluated as a valid diagnostic autopsy sign of premortem hanging as well as the proof of premortem hanging.
- Simon's sign can be considered as an objective vital finding, which is not specific for hanging. The absence of haemorrhages does not exclude death by hanging.
- Simon's haemorrhages have diagnostic significance in cases of hanging where there is a minimum number of findings on the cervical organs; in such situations, the presence of Simon's sign has high diagnostic value.
- Simon's sign in cases of hanging is more frequent in rather young individuals, in cases with free body suspension, and in individuals with minimal degenerative changes in the lumbosacral part of the spinal column.
- Simon's bleedings in cases of hanging most likely occur because of a combination of agonal convulsions and traction of the body as a result of gravity.
- Simon's sign in blunt trauma fatalities is most often connected with direct or indirect hyperextensive violence against the spinal column.
- Simon's bleedings in cases of natural death are exceedingly rare.

**Table 1.32 Frequency of Simon's bleedings as a vital sign of hanging**

| Author(s) | Investigated cases (*n*) | Positive cases (*n*) | Positive cases (%) |
|---|---|---|---|
| Simon (1968a) | 64 | 53 | 82.8 |
| Simon (1968b)[*] | 38 | 36 | 94.7 |
| Marcinkowski et al. (1972) | 17 | 11 | 64.7 |
| Geserick et al. (1976a) | 840 | 218 | 26.0 |
| | 43 | 20 | 46.5 |
| Geserick et al. (1976b)[**] | 58 | 25 | 43.1 |
| Saternus et al. (1979)[***] | 32 | 17 | 53.1 |
| Kleiber et al. (1982) | 222 | 104 | 46.8 |
| Braun and Toskos (2006) | 20 | 14 | 70.0 |
| Nikolic et al. (2009) | 147 | 93 | 63.3 |
| Hejna and Rejtarova (2010)[***] | 178 | 65 | 36.5 |
| Schulz et al. (2011) | 74 | 36 | 48.6 |

*Source:* Geserick G, Krocker K, Schmeling J. *Arch Kriminol* 2012;**229**:163–178.

[*] In this study the dorsal intervertebral disc was investigated.

[**] 840 cases were evaluated retrospectively, 43 cases were studied prospectively.

[***] The authors evaluated only bleedings of the lumbar spine.

## Respiration

In addition to reactions of the circulatory system, vital reactions of the respiratory system are also very important. These include acute pulmonary emphysema and aspiration/inhalation (e.g. in cases of neck compression, an increase in airflow resistance is observed). This is characterized by an inspiratory stridor. Due to an increase of $pCO_2$, an acceleration and deepening of the respiration is noted leading to acute pulmonary emphysema, and possibly also to interstitial emphysema. Acute pulmonary emphysema is also found in cases of drowning (Figure 1.36a,b). It loses its diagnostic value in cases of resuscitation with artificial ventilation or with putrefaction.

## Aspiration/inhalation

Aspiration of liquids and solids is an important sign of vitality in cases of aspiration of blood, soot, gastric contents, drowning, and so on. (Figure 1.37). Only inhalation into the deeper parts of the bronchial tree can prove vitality. Postmortem fluids might also flow passively into the trachea and the main bronchi (Table 1.33).

In cases of homicidal smothering with a pillow, the diagnosis of death by smothering may be difficult since external evidence of violence is scarce or even missing. In such cases, it may be important to search also for evidence of the aspiration of fabric fibres. Furthermore, the preservation of traces of fibre around the mouth and nose with adhesive tapes can demonstrate close contact of a pillow with the face, for example. Only the evidence of fibres in the trachea, however, can prove the vital obstruction of the airways by

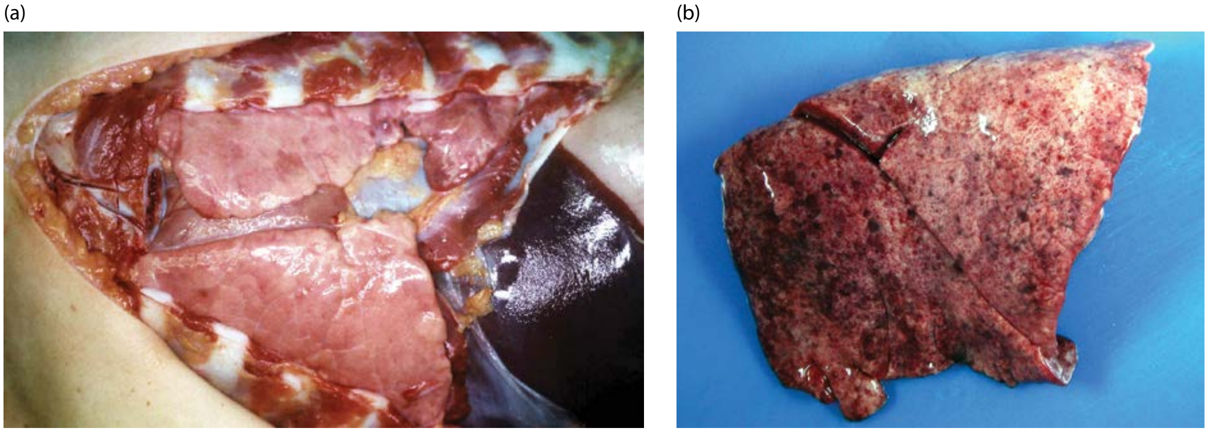

**Figure 1.36** (a) Massive pulmonary emphysema in a case of drowning. The margins of the lungs cover the mediastinum nearly totally (sometimes even overlapping). (b) Massive pulmonary emphysema in a homicide by drowning with numerous subpleural haemorrhages.

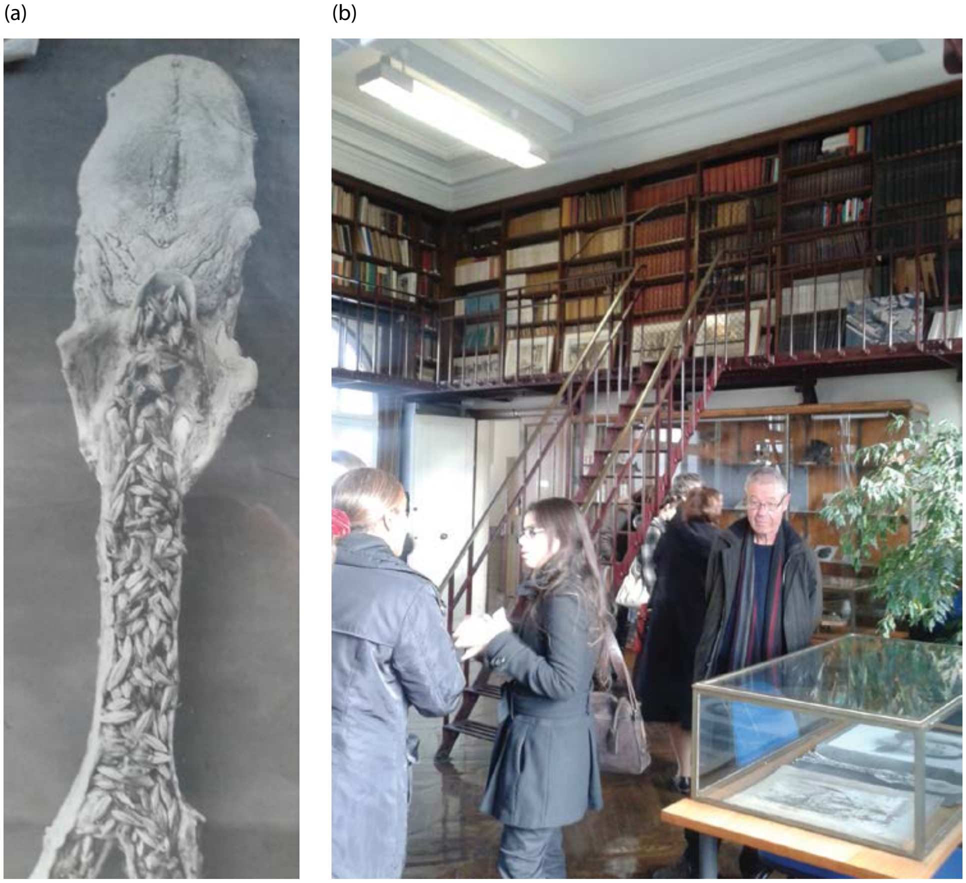

**Figure 1.37** (a) Aspiration of grain seed into the trachea and main bronchi. Original figure from the library of the Institute of Forensic Medicine in Paris. (b) Exhibit of the trachea is in the glass showcase in front of the author.

textile material and aspiration of fibres. This is illustrated by the following recent homicide case.

A slim 72-year-old woman was found dead in her living room. Autopsy revealed a compression of the thorax with contusion of the heart, and fracture of ribs 2 to 6 right and 5 and 6 left. Petechial haemorrhages were found in the eyelids, the conjunctivae, the face and the neck with, in addition, a bilateral hyposphagma. The cervical spine was injured between the fifth and the sixth vertebrae. On the arms, there were multiple haematomas. On the face, there were desiccated areas on the right front, on the left eyebrow, in the left nasolabial pleat and on the left cheek. On the lips, three superficial tears up to 5 mm in length were observed (Figure 1.38). These findings raised the

**Table 1.33 Vital reactions – aspiration/inhalation**

| Phenomenon | Mechanism | Detection | Postmortem origin |
|---|---|---|---|
| Inhalation of gas/fluids, penetration of liquids and solid particles into the respiratory tract during inspiration | | | In case of apnoea, only by insufflation (artificial ventilation) |
| Blood aspiration | 1. Bleeding penetrating into the tracheobronchial system<br>2. Ventilation | Macroscopic/ histological | In case of apnoea, only by insufflation/artificial ventilation |
| Brain tissue aspiration | 1. Head injury with basal skull fracture<br>2. Ventilation | Macroscopic/ histological | Spontaneous |
| Soot | 1. Soot production during fire/ smouldering fire<br>2. Ventilation | Macroscopic/ histological | If charring is extensive with charring defects to the trachea and the lungs, artificial findings are possible |
| Inhalation of hot gases | 1. Inhalation of hot air or steam<br>2. Thermic mucous membrane damage | Microscopic | In case of apnoea, only by insufflation; mucous membrane damage at least partly possible postmortem |
| Fluids (drowning) | 1. Submersion, originated in water<br>2. Ventilation | Macroscopic | Penetrating also possible for corpuscular components by hydrostatic pressure (depending on water depth) |

*Source:* Madea B, Grellner W, Kondo T. In: Madea B (ed.). *Handbook of Forensic Medicine*. Chichester, Wiley-Blackwell, 2014, pp 237–252.

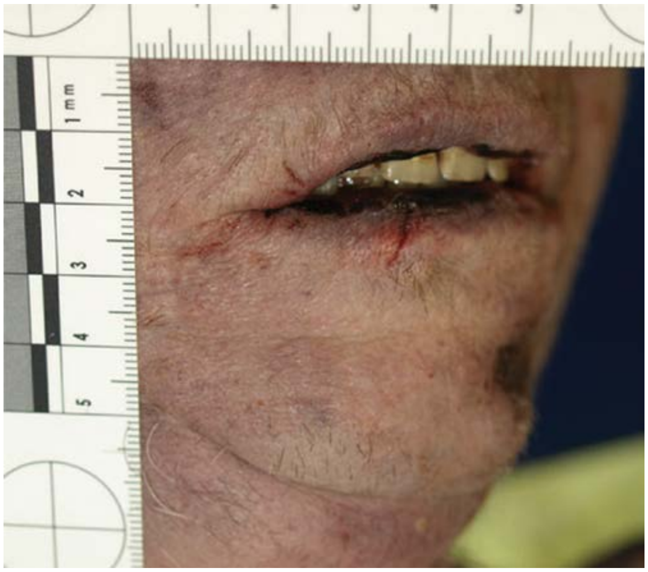

**Figure 1.38** Lips with superficial tears.

suspicion that a green pillow which was found at the scene had been pressed on the face. A tracheobronchial lavage with distilled water was therefore performed during autopsy. The about 50 ml (0.2 fl oz) liquid were centrifuged; the sediment was dashed out on slides and dried. Using a dissecting microscope at a magnification of ×45, several blue, green and cyan fibres were detected (Figure 1.39). The fibres were obviously aspirated during smothering.

Later, the murderer confessed that he had practised burking while smothering the woman with the green pillow. His motive was the theft of cash and the victim's bank card.

While fibres on adhesive tapes around the nose and mouth prove smothering, the finding of fibres in the trachea corresponds to a vital reaction and is evidence that tight contact with a fabric occurred during life. Therefore, a tracheobronchial lavage should be carried out in such cases.

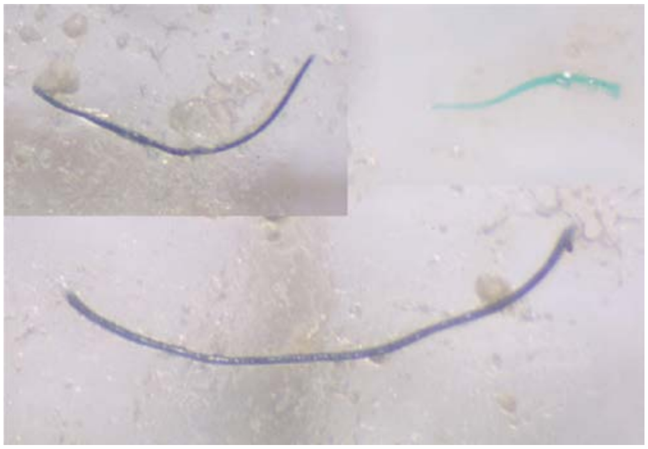

**Figure 1.39** Different fibres in the sediment of the tracheobronchial lavage (magnification ×45).

## Role of pulmonary macrophages and giant cells in fatal asphyxia

There is still controversial discussion on the issue of whether the appearance and number of macrophages and giant cells in pulmonary tissue can support the diagnosis and type of fatal asphyxia, in particular with regard to survival time (rapid or protracted asphyxia). Janssen (1963) and Janssen and Bärtschi (1964) described mobilization and proliferation of alveolar cells with the detection of giant cells under special conditions.

Betz et al. (1994) showed the presence of these cells also in other causes of death. Further investigations, including our own results, lead to different conclusions as to the role of pulmonary macrophages in asphyxia/suffocation.

Betz et al. (1993) studied the significance of pulmonary giant cells for the diagnosis of asphyxiation. Lung specimens of 54 individuals with different natural and unnatural causes of death were investigated. In most lungs examined, numerous alveolar macrophages with one or two nuclei were found. Polynuclear giant cells, which were arbitrarily defined as alveolar macrophages containing three or more nuclei, were observed in all groups investigated except in the cases of hypoxia due to covering the head with a plastic bag. Apparent differences between the other groups – an increased number in cases of throttling or strangulation – were not observed. Immunohistochemical investigations confirmed the hypothesis that the observed polynuclear giant cells were derived from alveolar macrophages. The immunohistochemical analysis of the proliferation marker antigen Ki-67 revealed no positive reaction in the nuclei of polynuclear giant cells, indicating that these cells had not developed shortly before death by endomitosis as an adaptive change following reduction in oxygen supply. The results provide evidence that the detection of pulmonary polynuclear giant cells cannot be used as a practical indicator for death by asphyxiation due to throttling or strangulation.

In a further study, a morphometric analysis was performed to elucidate the significance of pulmonary polynuclear giant cells as a histological sign of asphyxiation. A total of 13 cases of homicidal strangulation by throttling, 8 cases of traumatic asphyxia due to chest compression and 10 control cases (cause of death: severe head injury, no signs of aspiration or other relevant pulmonary alterations, smokers and non-smokers) were investigated. The number of alveolar macrophages containing one or two nuclei and of polynuclear giant cells per microscopic field was estimated and a statistical evaluation was carried out. A considerable individual variation was observed in all groups with a tendency to higher numbers of cells in cases of smokers or advanced individual age. However, no significant differences were detectable in the content of alveolar macrophages and of polynuclear giant cells between the asphyxiated individuals and the controls. Since polynuclear giant cells occurred in similar amounts in the healthy, functionally normal lungs of non-asphyxiated individuals, the detection of such cells cannot be regarded as a reliable indicator for asphyxiation.

According to Betz et al., the following practical conclusions for the morphological diagnosis of asphyxiation by detection of polynuclear giant cells can be drawn.

- Polynuclear giant cells can also occur in the lungs of healthy, non-asphyxiated individuals and considerable variations in the number of monoculear alevolar macrophages can be found.
- No significant increase in the number of alveolar macrophages or polynuclear giant cells can be observed in cases of strangulation/throttling or chest compression. The duration of the asphyxiation process, at least in most of these forensic autopsy cases, does not seem to be long enough to enable the development of pulmonary polynuclear giant cells as described in experimental animals with considerably longer episodes of hypoxia.

## Swallowing

Swallowing is defined as the voluntarily induced propulsory, reflectory, peristaltic transport of fluid or food into the stomach.

Swallowing of blood, foreign objects, tissue components, teeth, drowning fluid and soot occurs intravitally. However, drowning fluid found in the stomach alone is no evidence of drowning since water can get into the stomach even post mortem due to hydrostatic pressure. In cases of drowning, there is a typical separation of the gastric content into three phases, with a foamy phase on top, a liquid phase in the middle and a solid phase at the bottom (so-called Wydler's sign). Mucosal tears of the gastric mucosa that are caused by overexpanding of the stomach due to swallowed drowning fluid or to emesis under water against resistance are also vital reactions.

## Biochemical vital reactions and histology

For more than 70 years investigations have been carried out on various biochemical analytes as vitality markers, especially lactate, catecholamines and thyroglobulin (Table 1.34). Of note is Berg, who studied the suitability of catecholamines as vitality markers in strangulation (Berg and Bonte, 1973). In external asphyxiation, high catecholamine values were found in comparison to reflex death (Table 1.35). Furthermore, the catecholamine values are correlated with the duration of the terminal episode (Table 1.33). He claimed craniocaudal strangulation differences of the catecholamine concentration between left ventricular and sinus blood.

Kauert et al. (1990) carried out a follow-up study, analysing serum catecholamines in 26 cases of hanging, throttling, drowning and sudden unexpected death. Blood from the left and right ventricles and the sinus sagittalis superior was drawn in order to quantify adrenaline, noradrenaline and dopamine by high-performance liquid chromatography.

The results show that differences also exist in non-asphyxial death between catecholamine concentrations of right and left ventricular blood as well as of the left ventricular and sinus blood.

They conclude that causes other than cessation of flow must be considered:

**Table 1.34 Biochemical investigations in asphyxial deaths (according to Eisenmenger et al., 1990)**

| Parameter | Author | Year |
|---|---|---|
| Lactate | Swann and Brucer | 1949 |
| | Srch et al. | 1965 |
| | Sturner et al. | 1983 |
| Fatty acids | Gostomzyk and Frei | 1969 |
| | Sawaguchi et al. | 1974 |
| Hypoxanthine | Friedrich | 1986 |
| | Pietz et al. | 1988 |
| Phospholipids | Berg | 1952 |
| | Mueller | 1961 |
| | Laves and Berg | 1965 |
| | Weiler and Haarhoff | 1972 |
| | Döring | 1975 |
| | Saternus et al. | 1980 |
| | Haffner et al. | 1988 |
| Catecholamines | Berg | 1952 |
| | Ludemann et al. | 1955 |
| | Berg | 1963 |
| | Lund | 1964 |
| | Laves and Berg | 1965 |
| | Berg and Bonte | 1973 |
| | Kauert et al. | 1982 |
| | Yoshimoto et al. | 1984 |
| | Kauert | 1986 |
| | Kita | 1987 |
| Thyroglobulins | Yada et al. | 1971 |
| | Yada et al. | 1972 |
| | Yada et al. | 1973 |
| | Katsumata et al. | 1980 |
| | Katsumata et al. | 1984 |
| | Tamaki et al. | 1987 |
| | Müller and Franke | 1988 |
| Enzymes | Sawaguchi and Yoshinari | 1971 |
| | Sawaguchi | 1973 |
| | Friedrich | 1986 |
| Guanidine | Miyamoto | 1981 |
| Ascorbic acid | Arad et al. | 1985 |

- It is possible that the right–left ventricular differences are caused by the final release of adrenal catecholamines, which are not distributed any longer and/or by a pulmonary consumption of catecholamines.
- The differences between left ventricular and sinus blood may be caused by an agonal decreased cerebral perfusion leading to a predominant distribution of catecholamines in the body circulation.

The diagnosis of vital strangulation through differences in the concentration of biochemical parameters between head and body blood seems to be problematic.

Hirvonen et al. (1990) carried out animal experiments on stress hormones, which are rapidly released into blood during imminent asphyxia. They investigated stress hormone concentrations in plasma after two kinds of asphyxia in anesthetized rabbits: strangulation and $CO_2$ inhalation. Both adrenaline and noradrenaline concentrations rose manifold (eight and ten times respectively) in blood samples taken immediately after death by strangulation. In rabbits killed by an overdose of barbiturate, no rise in the levels of these hormones was found. Unfortunately, the concentrations were still higher in blood samples taken one- or two-days post mortem, thus hiding the initial increase caused by asphyxia. Blood cortisol and histamine did not change significantly during asphyxia.

The suitability of increased thyroglobulin (Tg) concentration for the diagnosis of vital strangulation was also investigated (Müller et al., 1990). Samples taken at medicolegal autopsies were centrifuged, the serum separated from erythrocytes and stored at 4°C. Tg was measured by radioimmunoassay. This method is an improvement on the method used by Yada et al. (1971), who demonstrated a qualitative test employing precipitation electrophoresis of thyroid extract with anti-Tg-serum. In their study, Müller et al. found a very high Tg value (more than 400 ng/ml Tg) in serum in all cases of throttling and manual strangulation (Table 1.36), but in cases of

**Table 1.35 Catecholamine concentration in cadaveric blood (serum in ng/ml) in 111 cases. Calculation of significance of pair differences (adrenaline–noradrenaline) according to Wilcoxon**

| Duration of terminal episode | Cause of death | Adrenaline | | Noradrenaline | | Difference | | |
|---|---|---|---|---|---|---|---|---|
| | | *m* | *s* | *m* | *s* | *A-NA* | *n* | *P* |
| I Short | External asphyxiation | 197 | 142 | 175 | 145 | 22 | 30 | 0.1 |
| | Internal asphyxiation | 105 | 73 | 134 | 119 | −29 | 12 | 0.1 |
| | Fatal haemorrhage | 128 | 143 | 98 | 141 | 30 | 12 | 0.05 |
| | Cardiac death | 135 | 88 | 195 | 148 | −60 | 16 | 0.1 |
| II Long | Cranio-cerebral trauma | 242 | 253 | 285 | 266 | −43 | 21 | 0.1 |
| | Intoxication | 200 | 127 | 129 | 108 | −29 | 10 | 0.1 |
| III Missing | Reflex death, bolus death, sudden cardiac death | 11 | 6 | 12 | 9 | −1 | 10 | |

*Source:* Berg S, Bonte R. *Z Rechtsmed* 1973;**72:**56–62.
*Note:* *m* = mean value.

**Table 1.36 Tg-serum concentration in 57 strangulation cases**

| Tg concentration (ng/ml) | Throttling/ligature strangulation ($n = 8$) | Hanging ($n = 49$) |
|---|---|---|
| <100 | – | 34 |
| 101–200 | – | 7 |
| 201–300 | – | – |
| 301–400 | – | 2 |
| >400 | 8 | 6 |

*Source:* Müller E, Eulitz J, Lobers W. In: Brinkmann B, Püschel K (eds). *Ersticken. Fortschritte in der Beweisführung*. Berlin, Springer Verlag, 1990, pp 64–69.

**Table 1.37 Suitability of different analytes for the diagnosis 'obstructive asphyxia' (according to Eisenmenger et al., 1990)**

| Analyte | Suitability |
|---|---|
| Lactate | 0 |
| Fatty acids | 0 |
| Hypoxanthine | 0 |
| Phospholipids | 2 |
| Catecholamines | 0 |
| Thyreoglobuline | 2 |
| Encymes | 1 |
| Ascorbic acids | To be checked |

*Note:* 0 = not suitable; 1 = suitability in question; 2 = conditional suitable; 3 = proof.

**Table 1.38 Validity of so-called vital signs in death due to strangulation**

| Vital signs | Valid |
|---|---|
| Petechial haemorrhages above the level of strangulation | √ |
| Haemorrhages of the neck | √ |
| Subperiostal haemorrhages of the clavicle | √ |
| Haemorrhages of the sternocleidoid muscle | √ |
| Haemorrhages of the skin between two ligatures | – |
| Anterior haemorrhages of the intervertebral discs | √ |
| Haemorrhages in the back | √ |
| Haemorrhages in auxiliary respiratory muscles | √ |
| Histamine content in the ligature mark | – |
| Haemorrhages of the intestinal wall | ? |
| Positive aquaporin-3 staining in skin | √ |
| Acute pulmonary emphysema | √ |
| Pulmonary macrophages and giant cells | – |
| Haemorrhagic dysoric syndrome | ? / – |
| Biochemical vital reactions: | |
| • Catecholamines | ? |
| • Thyroglobulin | √ |

hanging the concentration in serum varied greatly (from 12 to more than 400 ng/ml serum) in contrast to control cases of sudden death (Tg 22.7 ± 13.5 ng/ml). The reason for the different Tg concentrations is unknown. Müller et al. concluded that the duration of agony has an influence on the Tg concentration in cases of hanging. The highest concentrations were observed in cases of complete suspension of bodies. In a few autopsies involving CO poisoning and subarachnoid haemorrhage, high values of Tg were found. Müller et al. believed that this could be a result of chronic stress.

The suitability of different analytes for the diagnosis of 'obstructive asphyxia' is summarized in Table 1.37. A table with the validity of so-called vital signs and reactions in death due to strangulation can be found in Table 1.38.

## Vitality of strangulation marks

In forensic practice it is always important to determine the vitality of the strangulation mark. Conventional macroscopic and histological findings may be unreliable. The strangulation mark itself can also be produced post mortem or become even invisible after water exposure. Biochemical investigations on the histamine content of the strangulation mark have revealed no reliable results. Turillazzi et al. (2010) investigated the immunohistochemical expression of a panel of cytokines and inflammatory cells in skin specimens of autopsy cases of death due to hanging. They considered their significance in assessing whether the hanging mark and signs occurred before or after the death of the victim. An immunohistochemical investigation of skin samples was performed utilizing the antibodies anti-tryptase, fibronectin, TNF$\alpha$, IL-6, IL-8, IL-10, MCP-1, IL-15, IL-1$\beta$, CD45, CD4, CD3, CD8, CD68, CD20 and CD15. The authors concluded that tryptase, IL-15 and CD15 appeared to be reliable parameters in the determination of the vitality of ligature marks (Table 1.39).

The working group of Kondo (see also Chapter 15) investigated the suitability of the expression of aquaporin-3 and aquaporin-1 as vitality markers of strangulation marks (Ishida et al. 2018). Aquaporins are membrane water channels that play critical roles in controlling the water contents of cells. They are expressed in many epithelia and endothelia that can regulate osmolality throughout the body. Aquaporin-1 is expressed in vascular endothelial cells throughout the body, including the skin. Epidermal aquaporin-3 is increased in a cutaneous burn wound. A total of 24 neck compression marks by ligatures were investigated. After immunostaining, a total of ten high power fields were selected randomly. Antemortem neck compression enhanced aquaporin-3 expression in the keratinocytes. These results have been confirmed by this author's own working group (Figure 1.40). Aquaporin-3 might be useful as a marker of all kinds of antemortem skin injuries.

**Table 1.39** **Semi-quantitative evaluation and statistical analysis of the immunohistochemical findings and gradation of the immunohistochemical reaction in the marginal zones above and below the hanging marks**

| Antibody | A Soft marks 21 cases | B Hard marks 28 cases | C Controls 21 cases | | Statistical value |
|---|---|---|---|---|---|
| Fibronectin | – | – | – | | NS |
| | | | | A vs B | NS |
| | | | | A vs C | NS |
| | | | | B vs C | NS |
| Tryptase | ++++ | ++++ | – | | *** |
| | | | | A vs B | NS |
| | | | | A vs C | *** |
| | | | | B vs C | *** |
| IL-6 | + | + | – | | *** |
| | | | | A vs B | NS |
| | | | | A vs C | *** |
| | | | | B vs C | *** |
| IL-1β | – | – | – | | NS |
| | | | | A vs B | NS |
| | | | | A vs C | NS |
| | | | | B vs C | NS |
| IL-8 | + | + | – | | *** |
| | | | | A vs B | NS |
| | | | | A vs C | * |
| | | | | B vs C | *** |
| IL-10 | – | – | – | | NS |
| | | | | A vs B | NS |
| | | | | A vs C | NS |
| | | | | B vs C | NS |
| IL-15 | ++++ | ++++ | ++++ | | *** |
| | | | | A vs B | NS |
| | | | | A vs C | *** |
| | | | | B vs C | *** |
| TNF-α | – | – | – | | NS |
| | | | | A vs B | NS |
| | | | | A vs C | NS |
| | | | | B vs C | NS |
| CD45 (leucocyte common antigen) | +++ | +++ | +++ | | *** |
| | | | | A vs B | NS |
| | | | | A vs C | *** |
| | | | | B vs C | *** |
| CD3 (T-cell receptor complex) | + | + | – | | *** |
| | | | | A vs B | NS |
| | | | | A vs C | *** |
| | | | | B vs C | *** |
| CD4 (T helper cells | + | + | – | | *** |
| | | | | A vs B | NS |
| | | | | A vs C | *** |
| | | | | B vs C | *** |
| CD8 (cytotoxic T cells) | + | + | – | | *** |
| | | | | A vs B | NS |
| | | | | A vs C | *** |
| | | | | B vs C | *** |
| CD15 (neutrophilis) | ++++ | ++++ | – | | *** |
| | | | | A vs B | NS |
| | | | | A vs C | *** |
| | | | | B vs C | *** |

*(Continued)*

**Table 1.39 (*Continued*) Semi-quantitative evaluation and statistical analysis of the immunohistochemical findings and gradation of the immunohistochemical reaction in the marginal zones above and below the hanging marks**

| Antibody | A<br>Soft marks<br>21 cases | B<br>Hard marks<br>28 cases | C<br>Controls<br>21 cases | | Statistical value |
|---|---|---|---|---|---|
| CD20 (B-lymphocyte antigen | + | + | − | | *** |
| | | | | A vs B | NS |
| | | | | A vs C | *** |
| | | | | B vs C | *** |
| CD68 (macrophages) | + | + | − | | *** |
| | | | | A vs B | NS |
| | | | | A vs C | *** |
| | | | | B vs C | *** |

*Source:* Turillazzi E et al. Tryptase, CD15 and IL-15 as reliable markers for the determination of soft and hard ligature marks vitality. *Histol Histopathol* 2010;**25:**1539–1546.

*Note:* $p > 0.05$; *: $p < 0.05$; **: $p < 0.01$; ***: $p < 0.001$.

Intensity of immunopositive infiltrates were assessed semi-quantitatively in the scale 0–4 as follows:

−: no immunoreactivity (0%); +: mild immunopositivity in scattered cells (10%); ++: immunopositivity in up to a third of cells (33%); +++: immunopositivity in up to half of cells (50%); and ++++: strong immunopositivity in the majority or all cells (100%).

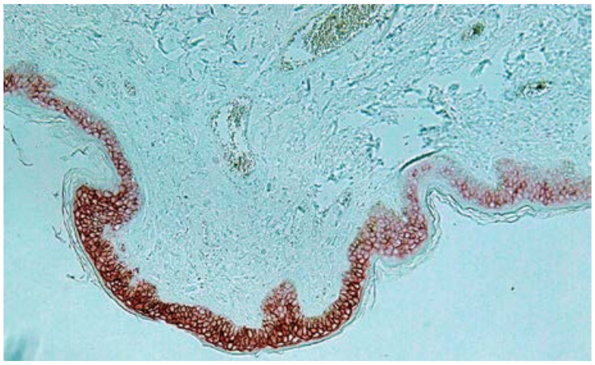

**Figure 1.40** Positive aquaporin-3 staining of the epidermis of a ligature mark.

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Since the bibliography is very long, and to support reading of the text, not all references have been cited in the text but are instead arranged here according to specific topics. References cited in the text sections are indicated by an asterisk (*).

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# 2 Nicolae Minovici and His Self-hanging Experiments

*George Cristian Curca*

To write the story of Nicolae Minovici (23 October 1868–26 June 1941) and his famous experiments of self-hanging is a tricky endeavour and a professional and historical challenge, not only because his were such strange medical experiments, but because the telling of the story must uncover and present an important personality of European legal medicine.

Nicolae Minovici (Figure 2.1) was the youngest son in his family. Mina Minovici, his elder brother, was an authoritative person. He became an eminent forensic pathologist who established the first Institute of Legal Medicine in Romania, which he directed for over 40 years. It was Mina who organized the legal medicine system in Romania as a public system, as it is today. Ten years younger than Mina, Nicolae Minovici loved the arts and projected a humanistic attitude towards everything surrounding him, particularly attentive to society needs. Mina Minovici asked him repeatedly to give up the humanistic ideals of his youth and to second him in his work as a forensic pathologist.

‘A dogged person, obstinate but determined’ as one of his professors characterized him, Nicolae Minovici finally accepted his elder brother’s offer and gave up his studies at Belle Arts Academy. He became a doctor in 1896 and wrote his PhD thesis in medicine in 1898 on ‘Tattoos in Romania’. He studied in Berlin from 1899 to 1901. Between 1901 and 1904 he developed a set-up for carrying out self-experiments on 172 hanging cases and in 1904 published his famous *Study on Hanging* [1] (Figure 2.2).

Nicolae’s interests were wide-ranging and he was determined that he could make improvements in many areas of society. In 1906 he set the foundations for the Ambulance Society in Bucharest, the first ambulance service in Romania. He was the promotor of the construction of the first emergency hospital in Bucharest (a landmark even today), part of his vision to create an emergency system in the capital, one of the few in Europe at that time.

In 1915 he was appointed associate professor of legal medicine and subdirector of the Institute of Legal Medicine in Bucharest built by his elder brother. In 1919 he set up the Institute of Legal Medicine in Cluj-Napoca, the largest city in the Transylvania region of the country.

In 1926 he was appointed mayor of the poorest area of Bucharest, the blue district.

He then became frenetically engaged in social battles. He fought against begging, but not against beggars, by setting up an ambitious social project to improve their lives, giving them work and creating a canteen where all poor people could have a warm meal every day. He also fought against occultism and obscurantism, and as President of the National College of Doctors was demanding severe penalties for those who usurped a doctor’s authority or initiated suspect treatments which reduced people’s chances of having their health problems resolved. He campaigned for the building of public toilets in Bucharest, the demolition of decaying houses and miserable neighbourhoods and the construction of habitable homes (‘Minovici demolish all, Minovici is a pickax’) [2]. Finally, he gave his own money to poor people and orphaned children and supported bright pupils from rural areas without financial means to study in Bucharest. A second appointment was as mayor of the entire region surrounding the city, a result of encouragement and pressure from the local people and community.

Minovici sought to protect traditional values and culture in Romania and he established the first museum of popular art in his own home. In the last years of his life, he bequeathed his beautiful villa and its entire surrounding parkland to the city of Bucharest (Figure 2.3). This was to become the Museum of Popular Art Dr Nicolae Minovici, where all the original popular and traditional artefacts he gathered from all over the country are presented for new generations in a huge collection today – one of Romania’s national treasures [2].

In his career, Minovici was responsible for the formation of the Romanian Society of Legal Medicine as the National professional body, of the *Journal of Legal Medicine*, the first national journal in legal medicine, the official publication of the Society. The journal continues today, known since 1993 as the *Romanian Journal of Legal Medicine*, an indexed ISI Thomson Reuters journal in the eastern part of Europe. Minovici wrote over 100 original scientific papers in various medical journals, gaining a scientific authority widely recognized in western countries with his name cited in connection with his scientific work and his self-hanging experiments.

Following the death of his brother Mina in 1933, Professor Nicolae Minovici, became full Director of the Institute of Legal Medicine in Bucharest for the following six years to come. In 1937, he was the secretary of the XVII Congress of International Anthropology and Prehistorian Anthropology held in Bucharest, Romania and of the VII General Assembly of the International Institute of Anthropology, successfully scientific meeting at that

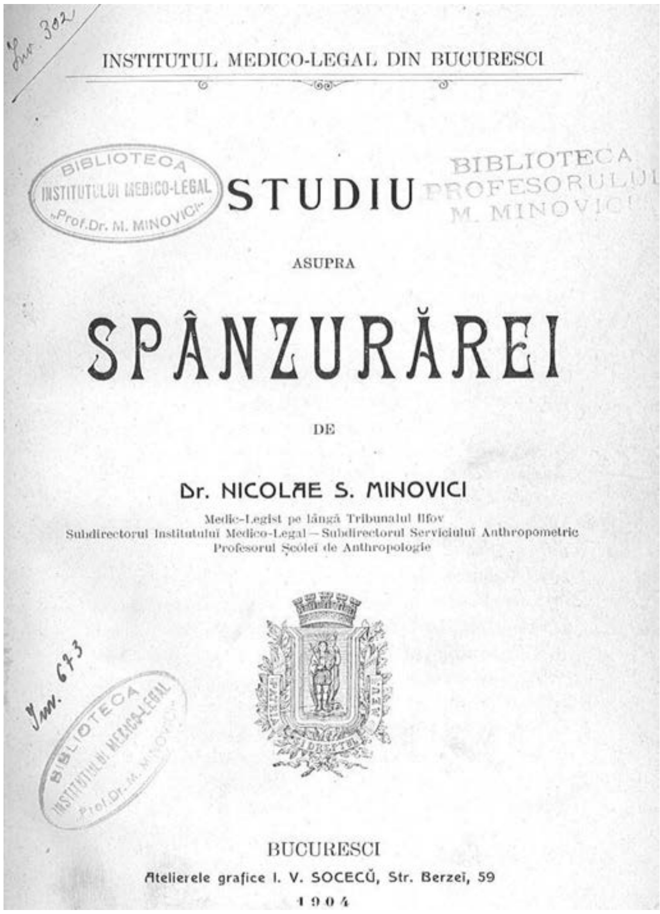
**Figure 2.1** Nicolae Minovici.

time which gathered many renowned scientists from 17 countries including the USA, UK, Mexico, India etc. under the presidency of Louis Martin and Eugene Pittard. Unfortunately, he was diagnosed with laryngeal cancer in 1938, probably a result of his famous but traumatic experiments of self-hanging around 30 years before, and he died in 1941 [2].

Professor Mina Minovici used to say that forensic pathologists are specialists on asphyxia because of the high frequency of such cases and because hypoxia is a mechanism in a variety of other causes of death, not just mechanical asphyxia. In 1904 Nicolae Minovici wrote:

> 'While in the western countries, where suicide is to be found more frequent in rural areas, we in our country we have more cases in cities where a lot of people choose to hang themselves. Individuals who apparently lack means to sustain life choose their own death. Large cities offer to those persons determinant causes to commit suicide by hanging' [3].

Nicolae Minovici had a fierce scientific curiosity and was keen to find answers on the subject of suicide, which he observed was a major area of the forensic specialty. The questions asked about strangulation and hanging at that time regarded the differentiation between hanging and ligature strangulation of the neck and also the differentiation between intra-vitam and postmortem hanging. The young forensic scientist was intrigued to



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Medic-Legist pe lângă Tribunalul Ilfov
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**Figure 2.2** Study on hanging.

**Figure 2.3** Nicolae Minovici museum of popular art.

find out the mechanisms of asphyxia by lack of air or by suppressing the blood supply needed for brain function.

In an oleograph report in 1901, Emil von Behring, Nobel Prize laureate in the same year, argued to the Neisser case (a case examining patient consent) that, ethically, self-experimentation needed always to precede experiments on human subjects. Emil von Behring and many other scientists believed that human subject experimentation without prior voluntary and informed consent was unethical.

In this more ethically aware scientific atmosphere post-Neisser, and especially bearing in mind the authoritative words of von Behring, Dr Minovici took the decision to set up a self-hanging experimental method in systematic research into hanging. This was a new approach and one that was extremely courageous given how dangerous these experiments were; they put life in peril and had therefore only been performed on animals previously. He carried out research on hanging by subjecting himself to self-experimentation, maybe the only scientific study of hanging on human subjects.

In his book, Minovici wrote [3]:

> ‘This study is not improvised and is the fruit of a hard work and medico-legal practice for several years. We aimed to find out what is not yet found in legal medicine on these themes or at least to reduce or to eliminate what is unneeded. Additionally, to encourage all those which are interested to discuss our study to present their remarks in order to make altogether a solid work.
>
> ‘While resting on the bed facing up and set on a pillow, I compressed with my fingers oriented to the vertebral column the neck vessels at the level of the hyoid bone and larynx. In less than 4–5 seconds we feel that a vail is falling down on us before our eyes, the sight is becoming foggy and eventually blackens. If we take off the fingers compression, we may feel like a burn sensation and a weight that spreads into our body from the occipital region to the toe fingers as paresthesia or even better described as an electric discharge.
>
> ‘The first time when I tried to kill myself in this way, I was lying on my bed watching the sky. With both hands, I compressed my neck around Adam’s knot. In the fifth second, I felt a purple vail coming down on me and then a black one. Amazingly fast, before my own eyes, images from my life marched as icons. Then I felt a huge electrical discharge, and everything turned black. I lost sense of ration.’

Minovici’s next step was to experience an incomplete hanging, using a rope of 5 mm thickness (Figure 2.4) with a loose knot connected to a dynamometer and a traction mechanism. He noted that he lost consciousness very soon, in 5–6 seconds, with 25–30 kg on the dynamometer (Nicolae Minovici’s weight was 69 kg). He noticed that when the knot was at the occiput, the loss of consciousness was extremely fast, with the knot laterally on the neck, the loss of consciousness followed after 8–9 seconds. He appreciated that even 5 kg was enough to cause complete cerebral hypoxia.

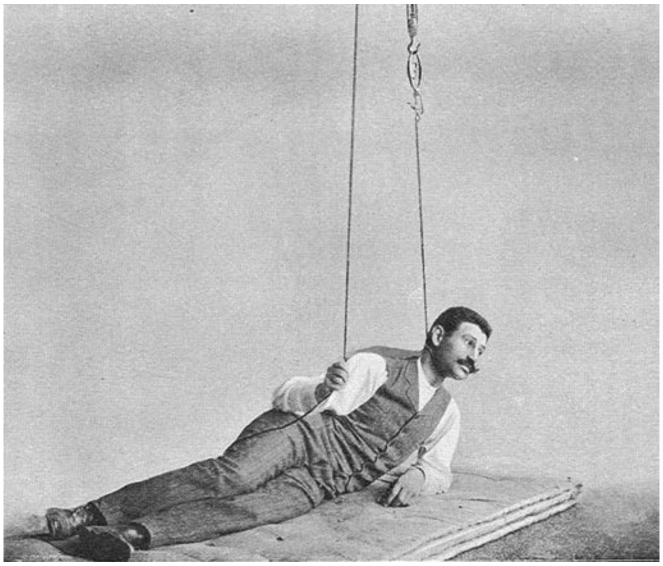

**Figure 2.4** Incomplete hanging.

The final step was to perform a complete hanging. He chose to use a 4 cm wide twisted rope made from a sheet and put the anterior bail of the rope next to the hyoid bone and the posterior bail of the rope next to the mastoid bones. He then proceeded to make six or seven complete hangings for just 4–5 seconds while his entire body was lifted 1–2 metres from the ground. He proceeded to carry out a total of 12 hangings of no more than 26 seconds each (Figure 2.5).

Minovici noted an intense pain and constriction in the hyoid bone immediately after his feet were lifted from the ground. After the procedure was finished, he experienced intense pain on swallowing (deglutition) every time. The maximum duration of the hanging was 26 seconds. Figure 2.5 shows the moment when his 70 kg were lifted to 1 metre for 18–20 seconds. He noted [3]:

> ‘From the first moments, the eyelids were so heavy that I couldn’t open the eyes, airways were completed collapsed. I could not hear my assistant voice numbering further than 5–6. My eyes were in tears and my ears ringing. I could not eat or drink normally even 12 days after. I was continuously thirsty 1–2 days without stop having my pharyngeal mucous intensely hyperemic. The hanging groove appears on my neck after 5–10 minutes and lasted until 8–11 days.
>
> ‘I tried to make a fluent knot on the rope to convolute my neck and to simulate a complete hanging while my body was completely lifted from the ground; I confess that despite my courage I

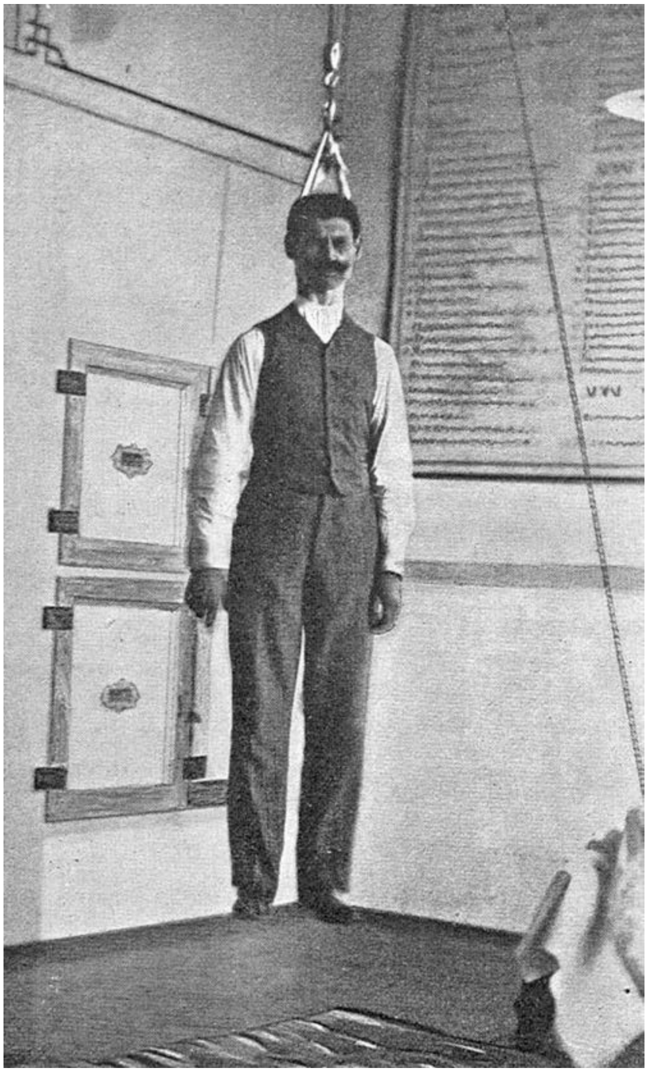

**Figure 2.5** Complete hanging.

couldn't go on with this experience for more than 3–4 seconds because the constriction and the pain I felt in my neck was so intense even before lifting up my body so I had to abort making conventional signs to my assistants. This last complete hanging produced me an intense pain in the larynx and hyoid bone and severe deglutition difficulties for one month. I can make a conclusion that in incomplete hanging blood vessels obliteration is far more important than airway obliteration in the mechanisms of death. When I was in lateral position my face was bluish but the blood still flows through the carotid oriented to the knot.

Another conclusion is that blood vessels compression ends life before vagus nerve could have an effect on the mechanisms of death.'

In the 136 cases of which he had personal professional experience, he observed that the heart always contains blood clots when the autopsy is made shortly after death.

In this book Minovici gives short answers from his experience to several important legal medicine questions:

- Was the hanging vital or postmortem? He insists that the pathologist must take into consideration all the external and internal signs and that there is none which is solely pathognomonic for a correct answer.
- Are the hanging and asphyxia the cause of death? A thorough external and internal examination of the signs of asphyxia and particular aspects of the groove may be conclusive.
- Is a suicide an accident or a homicide? In a suicide, the death scene usually is conclusive; in accidents there are more frequent sexual involvements and, in a homicide, there are often other traumatic lesions to be found.

He concluded that:

> 'The study of hanging opens a vast area of research enough to fulfill the scientific curiosity and professional duties, practically unlimited. We are far away to believe that this subject is closed, or science has concluded his final word or to drop of any further study. There is no doubt that a solution to this charade will be found soon. Human mind imagines today what yesterday appeared to be only fantasies. Science has now gotten into a world of visions and what seems to be impossible today, tomorrow will be pure reality' [3].

Nicolae Minovici's experiments of self-hanging represent an altruist offering to science from a man who worked tirelessly for the benefit of both science and society, to an extent that has scarcely been repeated.

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# 3 Crime Statistics

*Burkhard Madea*

*Homicide* is defined as the act of one human killing another. Homicides can be divided into many overlapping legal categories such as murder, manslaughter, justifiable homicide, killing in war, euthanasia and capital punishment.

Criminal homicide takes many forms, including accidental or purposeful murder [1,3,6,7–10,12–15,19,21,25,26,27–34,36].

Murder is the most serious crime. Although categories of murder can vary by jurisdiction and from country to country, murder charges fall into two broad categories:

- *First-degree murder* – the premeditated, unlawful, intentional killing of another person.
- *Second-degree murder* – the intentional, unlawful killing of another person, but without any premeditation.

In contrast, manslaughter is a form of homicide in which the person who commits the homicide either does not intend to kill the victim, or kills the victim as the result of circumstances that would cause a reasonable person to become emotionally or mentally disturbed to the point of potentially losing control of their actions.

The two broad categories of manslaughter are:

- *Voluntary manslaughter* – the intentional, killing of another person, but without premeditation and as the result of *a disturbed state of mind, or heat of passion.*
- *Involuntary manslaughter* – the unintentional killing of another person through an act of recklessness that shows indifference to the lives and safety of others, or an act of negligence that could reasonably be foreseen to result in the death.

The United Nations Office on Drugs and Crime (UNODC) has prepared a Global Study on Homicide before 2011 [20]. Qualifying intentional homicide, it is clear that the label ‘intentional homicide’ includes a very wide range of acts, not all of which are necessarily similar, other than the fact that they can be essentially represented by one person intentionally inflicting death on another person. As such, an examination of the ‘phenomenon’ of homicide requires significantly more than examination of overall ‘total homicide’ statistics. One challenge is to identify the most appropriate way in which to divide, or disaggregate, acts of homicide in order to generate meaningful subcategories that remain open to cross-national examination. Such categories may include the characteristics of the victim, the characteristics of the perpetrator, the nature of the offender–victim relationship, the weapon used, the physical location of the event, the time of the event, aggravating factors such as the involvement of drugs or alcohol, the motive, and the involvement of elements such as organized criminal groups.

A recent work on the classification of homicide [20], recognizes the need for homicide data disaggregation and proposes that many such elements could be considered as ‘horizontal attributes’ to be applied to all events fall within the general category of ‘intentional homicide’. As shown in Table 3.1, these attributes may be grouped in five general clusters, i.e. mechanism, victim/perpetrator attributes, victim/perpetrator relationship, geographic/location attributes, and situational context. Information on each of these aspects is available to different extents. Efforts are needed to standardize the recording of such characteristics both within and between countries in order to produce meaningful statistical data.

**Table 3.1 Disaggregating homicide statistics***

| Mechanism | Victim and perpetrator attributes | Victim/perpetrator relationship | Geographical/ location attributes | Situational context |
|---|---|---|---|---|
| • Use of weapon<br>• Firearm<br>• Knife<br>• Blunt object<br>• **Strangulation**<br>• Etc. | • Male/female victim<br>• Male/female perpetrator<br>• Child perpetrator<br>• Victim under influence of drugs/ alcohol<br>• Perpetrator under influence of drugs/ alcohol<br>• Etc. | • Intimate partner perpetrator<br>• Perpetrator related to victim<br>• Perpetrator known to victim<br>• Perpetrator unknown to victim<br>• Etc. | • Urban/rural<br>• Private residence<br>• Commercial property<br>• Street<br>• Other public places<br>• Etc. | • Organized crime-related<br>• Gang-related<br>• Robbery/ theft-related<br>• Intimate partner/ family-related<br>• Etc. |

* Reproduced from Injury Prevention. In: Epidemiology of violent deaths in the world Reza A et al (eds), 2001, p106 with permission from BMJ Publishing Group Ltd.

**Table 3.2** **The top five countries with the highest homicide rates from each of five continents in 2004. Rates are per 100 000 population**

| Africa | America | Asia | Oceania | Europe |
|---|---|---|---|---|
| South Africa<br>39.5–69.0 | Colombia<br>45.5–61.1 | Philippines<br>11.9–21.0 | Papua New Guinea<br>15.2 | Russian Federation<br>18.9–29.7 |
| Côte d'Ivoire<br>45.7 | El Salvador<br>56.4–57.5 | North Korea<br>18.9 | Nauru<br>9.9 | Ukraine<br>8.0–12.0 |
| Lesotho<br>13.3–37.3 | Jamaica<br>33.7–55.2 | Cambodia<br>3.7–18.5 | Kiribati<br>6.5 | Belarus<br>8.3–10.2 |
| Burundi<br>35.4 | Venezuela<br>32.5–37.0 | Kazakhstan<br>11.9–16.2 | Fiji<br>0.7–2.8 | Republic of Moldavia<br>7.2–8.2 |
| Congo<br>35.2 | Bolivia<br>32.5–37.0 | Myanmar<br>15.7 | Marshall Islands<br>1.8 | Turkey<br>2.9–6.9 |

*Source:* Data © United Nations Office on Drugs and Crime International Homicide Statistics [35].

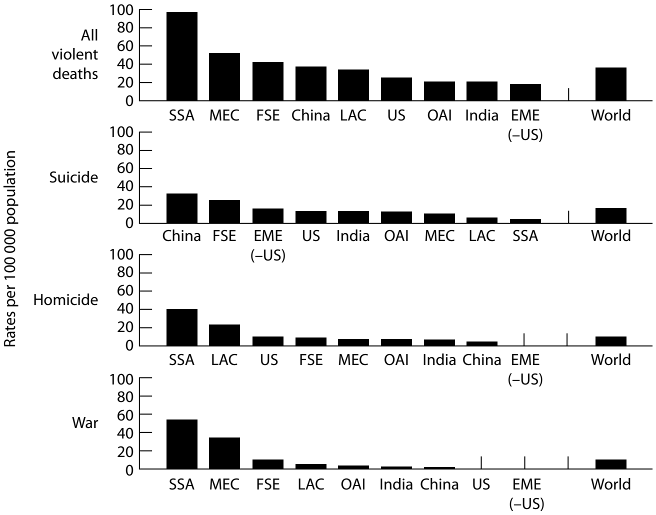


**Figure 3.1** Rates of violence-related deaths by region, 1990 (from Reza et al. [25]). Key: EME (US) = established market economies excluding the United States; FSE = formerly socialist economies; LAC = Latin America and the Carribean; MEC = Middle Eastern crescent; OAI = other Asia and islands; SSA = Sub-Saharan Africa.

According to the United Nations study of 2011 [20], the total number of homicides globally for 2010 was estimated at 468 000. More than one third (36%) occurred in Africa, 31 per cent in the Americas, 27 per cent in Asia, 5 per cent in Europe and 1 per cent in Oceania (Table 3.2). While the homicide rate has been falling since 1995 in Europe, North America and Asia, it has risen in Central America and the Caribbean. Of all homicide victims worldwide, 82 per cent were men and 18 per cent women. The homicide rate in Africa and the Americas (at 17 and 16 per 100 000 population respectively) is more than double the global average (6.9 per 100 000), whereas in Asia, Europe and Oceania (between 3 and 4 per 100 000) it is roughly half.

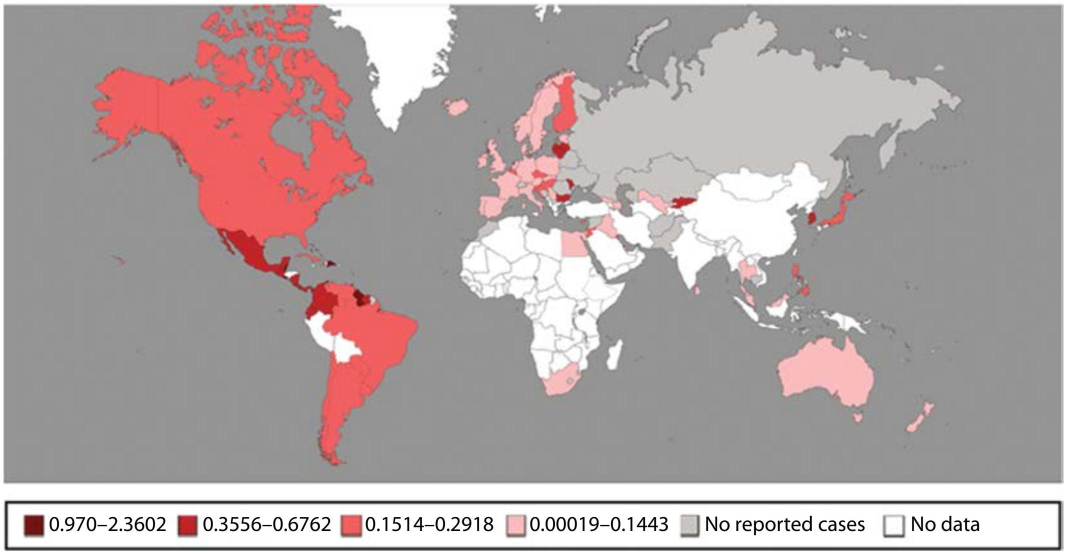
According to a 2013 UNODC report [35], the homicide rate dropped to 437 000 in 2012. The Americas accounted for 36 per cent of all homicides globally, Africa 21 per cent, Asia 38 per cent, Europe 5 per cent and Oceania 0.3 per cent. About 41 per cent of worldwide homicides in 2012 resulted from the use of guns, 24 per cent from sharp objects such as knives, and 35 per cent by other means such as a poison.

A review of the epidemiology of violent death in the world revealed that in 1990 an estimated 1 851 000 people died from violence (35 per 100 000), representing 3.7 per cent of all deaths occurring in the world that year (Table 3.3). Suicide was the most frequent form of violent death, followed by homicide and then war-related deaths [22]. There were an estimated 786 000 suicides. Overall suicide rates ranged from 3.4 per 100 000 in Sub-Saharan Africa to 30.4 per 100 000 in China. There were an estimated 563 000 homicides. Overall homicide rates ranged from 1.0 per

**Table 3.3** **Percentage of all deaths that were violence related, 1990 (according to Reza et al. [25])**

| | | Percentage of deaths due to violence | | | |
|---|---|---|---|---|---|
| **Country/region** | **Total deaths from all causes** | **Overall violence (%)** | **Suicide (%)** | **Homicide (%)** | **War (%)** |
| Established market economies excluding United States | 7 121 000 | 1.2 | 1.1 | 0.1 | 0.0 |
| Formerly socialist economies | 3 791 000 | 3.7 | 2.1 | 0.8 | 0.8 |
| India | 9 371 000 | 1.7 | 1.1 | 0.6 | 0.0 |
| China | 8 885 000 | 4.4 | 3.9 | 0.6 | 0.0 |
| Other Asia and islands | 5 534 000 | 2.4 | 1.2 | 0.9 | 0.3 |
| Sub-Saharan Africa | 8 202 000 | 6.0 | 0.2 | 2.5 | 3.3 |
| Latin America and Caribbean | 3 009 000 | 4.8 | 0.7 | 3.4 | 0.6 |
| Middle Eastern crescent | 4 553 000 | 5.6 | 1.0 | 0.9 | 3.7 |
| United States | 2 148 000* | 2.7 | 1.4 | 1.2 | 0.0 |
| World | 50 467 000 | 3.7 | 1.6 | 1.1 | 1.0 |

* *Source:* National Center for Health Statistics. Table 6. Deaths and death rates for the 10 leading causes of death in specified race–sex groups: United States, 1990. *Monthly Vital Statistics Report* 1993;**41**(7 suppl)**:**20.



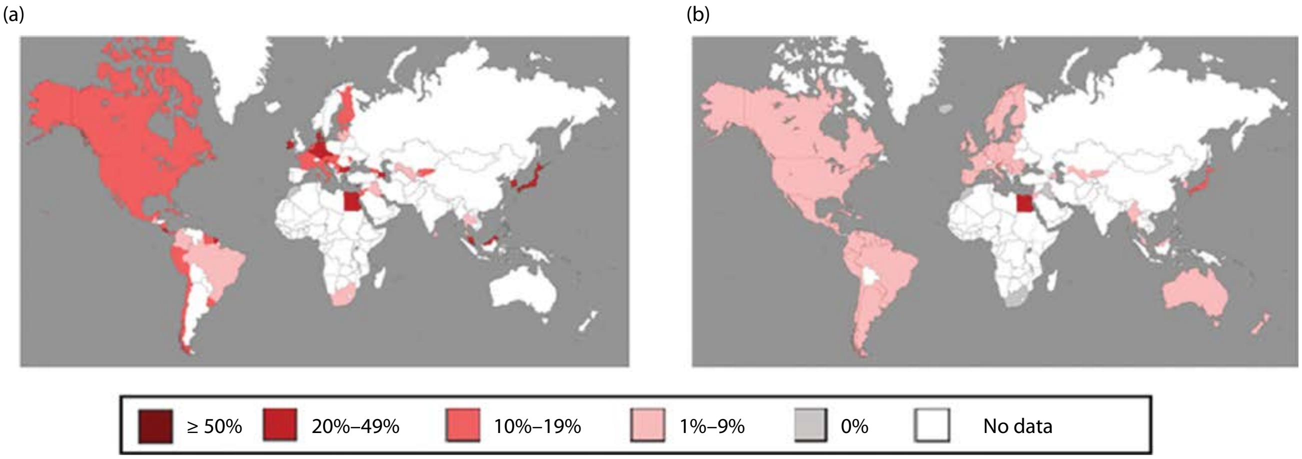
*Note*. Rates shown are the average of up to five of the most recent years of data available for each country. Data were obtained from the World Health Organization's Detailed Mortality Database. Mortality, *ICD-10*. Available at: http://www.who.int;healthinfo/statistics/mortality rawdata/ en/index.html. Accessed 14 February 2013.

**Figure 3.2** Homicide by asphyxiation, per 100 000 women aged ≥15 years old (from Sorenson et al. [31]).

100 000 in established market economies to 44.8 per 100 000 in Sub-Saharan Africa, with peaks among males aged 15–24 years, and among females aged 0–4 years. Suicide rates were highest in China and formerly socialist economies, homicide rates were highest in Sub-Saharan Africa and Latin America/Caribbean, and war-related death rates were highest in Sub-Saharan Africa and the Middle Eastern crescent (Figure 3.1). Limitations in available data point to the need for broader implementation of surveillance systems for violent death and injury. A global strategy is needed to address the premature and unnecessary deaths and disabilities associated with violence.

According to Sorenson et al. [31], strangulation is a relatively common cause of homicide death, particularly for women. The authors compiled data available through the World Health Organization to document the risk of homicide by asphyxiation among women around the globe and to illustrate gender differences in the risk of homicide by asphyxiation. As shown in Figure 3.2, rates vary widely, and many countries, particularly low-income countries, report no cases or have no data.

Figure 3.3 documents that asphyxiation accounts for a higher percentage of homicide of women than of men. These mortality data are parallelled by self-reported data.



*Note*. Rates shown are the average of up to five of the most recent years of data available for each country. Data were obtained from the World Health Organization's Detailed Mortality Database. Mortality, *ICD-10*. Available at: http://www.who.int;healthinfo/statistics/mortality rawdata/ en/index.html. Accessed 14 February 2013.

**Figure 3.3** Percentage of homicides by asphyxiation among those aged ≥15 years old by (a) women and (b) men (from Sorenson et al. [31]).

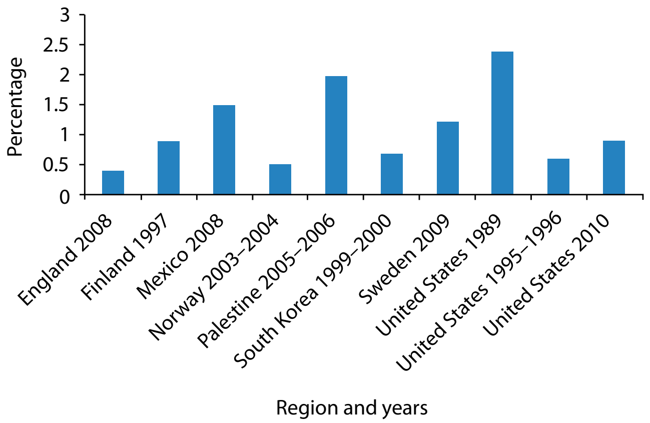


**Figure 3.4** Strangulation by an intimate partner, 1-year prevalence, women (%) (from Sorenson S, Joshi M, Sivitz E. *Am J Public Health* 2014;**104**(11):e54–e62).

Moreover, six times as many homicides of women (vs. men) are by an intimate partner.

The systematic review of the epidemiology of non-fatal strangulation by Sorenson et al. [31] revealed the following:

- Past-year strangulation victimization rates ranged from 0.4 per cent to 2.4 per cent for women (mean = 1.1%; median 0.9%; Figure 3.4). Lifetime victimization rates ranged from 3.0 per cent to 9.7 per cent (mean = 5.7%; median = 5.5%). The four studies that assessed past-year victimization of men found that women are two to four times as likely as men to report having been strangled by an intimate partner; the lifetime discrepancy increased to between 4- and 11-fold.
- Among women, 5-year strangulation victimization was higher if they were cohabiting (0.8% vs 0.3% if married), in a step-family (1.5% vs 0.5% if biological family), disabled (0.6% vs 0.3% if non-disabled), a renter (0.7% vs 0.3% if homeowner), or lived outside Quebec (0.4% vs 0.3% if lived in Quebec).

According to this survey, the prevalence of strangulation appears to have decreased in Canada, the only country with multiple cross-sectional surveys that have measured strangulation.

A review of asphyxiation homicides of 59 women in Norway and Denmark documented that a majority of women were manually strangled and then strangled with a ligature during the fatal incident [26]. Case files of 106 men who murdered a female intimate in England, Wales and Scotland indicated that 29 per cent of the women died as a direct result of the strangulation and another 8 per cent were strangled during the assault but died of another cause [11]. Thus, external cause of death numbers do not necessarily reflect the nature or scope of the use of strangulation in homicide.

In addition, few nations have crime or other databases that document both the method of death and the victim/perpetrator relationship, so individual research studies are necessary to understand the nature and scope of such mortality. Furthermore, a large case-control study in the United States found previous strangulation to be a substantial and unique predictor of attempted and completed homicide of women by a male intimate partner [15].

An example of how, in Hong Kong and Germany, the severity of injuries inflicted is directly linked to criminal liability and also the punishment is given in Table 3.4.

*Suicide* is an act of intentionally causing one's own death [23]. Approximately 0.5–1.4 per cent of people die by suicide, about 12 per 100 000 persons per year. Globally, suicide resulted in 828 000 deaths in 2015 (up from 712 000 deaths in 1990). This makes it the tenth leading cause of

**Table 3.4 An example of how, in Hong Kong and Germany, the severity of injuries inflicted is directly linked to criminal liability and the punishment imposed for the crime (according to Beh [3])**

| Laws of Hong Kong (HK) | Laws of Germany | Offence | Penalty as stated in law (HK) | Penalty as stated in law (Germany) | Penalty in reality (HK) |
|---|---|---|---|---|---|
| Chapter 212, Section 2 | § 211 | Murder | Life imprisonment | Life imprisonment | Life imprisonment |
| Chapter 212, Section 7 | § 212 | Manslaughter | Up to life imprisonment | Up to life imprisonment | Several years the norm. Life is rare. |
| Chapter 212, Section 212 | § 217 | Infanticide | Up to life imprisonment | Up to life imprisonment | Suspended sentence or care order. Rarely imprisonment. |
| Chapter 212, Section 19 | § 226 | Assault causing grievous bodily harm | Up to 3 years | 1–10 years | Months to 3 years |
| Chapter 212, Section 39 | § 223 | Assault causing actual bodily harm | Up to 3 years | Up to 5 years | Usually months |
| Chapter 212, Section 40 | § 224 | Common assault | Up to 1 year | 6 months to 10 years | Fines and suspended sentence not uncommon |

*Source:* Beh P. In: Madea B (ed.). *Handbook of Forensic Medicine*. Chichester, Wiley-Blackwell, 2014, pp 207–210.

Table 3.5 Suicide by method (% out of all suicides by country) according to the WHO Mortality Database (as of November 2006) [36]; countries reporting *ICD-10* data [37]

| Country | Years | Men | | | | | | | | Women | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Other poisoning | Pesticides | Hanging | Drowning | Firearms | Falls | Other | *N* | Other poisoning | Pesticides | Hanging | Drowning | Firearms | Falls | Other | *N* |
| **Africa** | | | | | | | | | | | | | | | | | |
| South Africa (ZA) | 1996 & 2004 | 6.6 | 3.6 | 68.7 | 0.0 | 12.6 | 0.2 | 8.2 | 412 | 22.7 | 12.6 | 41.2 | 0.8 | 9.2 | 0.8 | 12.6 | 119 |
| **Americas** | | | | | | | | | | | | | | | | | |
| Argentina (RA) | 1997–2003 | 0.7 | 1.7 | 49.1 | 1.5 | 37.6 | 2.4 | 7.0 | 15 214 | 3.4 | 4.1 | 38.0 | 4.2 | 25.9 | 10.3 | 14.1 | 4 188 |
| Brazil (BR) | 1996–2002 | 2.0 | 8.3 | 52.4 | 0.9 | 22.1 | 1.8 | 12.6 | 33 072 | 6.5 | 16.0 | 37.6 | 2.3 | 13.4 | 3.9 | 20.4 | 8 591 |
| Canada (CDN) | 2000–2003 | 10.2 | 0.4 | 44.4 | 2.3 | 21.6 | 4.7 | 16.3 | 11 419 | 34.3 | 0.5 | 36.8 | 4.0 | 3.8 | 6.5 | 14.1 | 3 288 |
| Chile (RCH) | 1997–2003 | 0.6 | 5.0 | 77.2 | 0.9 | 11.7 | 0.7 | 3.9 | 7 995 | 7.7 | 9.8 | 62.6 | 2.7 | 8.0 | 2.0 | 7.2 | 1 342 |
| Colombia (CO) | 1997–1999 | 5.9 | 20.1 | 27.7 | 1.1 | 37.0 | 3.1 | 5.1 | 4 243 | 12.5 | 45.7 | 17.4 | 1.2 | 15.0 | 4.2 | 3.9 | 1 292 |
| Costa Rica (CR) | 1997–2004 | 3.5 | 29.9 | 38.4 | 0.2 | 24.0 | 1.6 | 2.4 | 1 727 | 8.9 | 43.2 | 30.4 | 0.4 | 11.3 | 3.9 | 1.9 | 257 |
| Cuba (CU) | 2001–2004 | 1.7 | 8.9 | 76.8 | 0.6 | 3.4 | 2.0 | 6.6 | 4 620 | 11.5 | 10.3 | 27.4 | 1.3 | 0.7 | 3.0 | 45.8 | 1 649 |
| Dominican Republic (DOM) | 1996–2001 | 2.5 | 22.4 | 42.8 | 2.5 | 20.2 | 1.5 | 8.1 | 754 | 7.8 | 34.9 | 31.9 | 3.6 | 8.4 | 3.0 | 10.2 | 166 |
| Ecuador (EC) | 1997–2004 | 1.6 | 32.2 | 41.3 | 1.6 | 19.2 | 0.1 | 4.0 | 3 369 | 2.3 | 64.3 | 23.9 | 0.8 | 5.3 | 0.1 | 3.3 | 1 542 |
| El Salvador (ES) | 1997–2003 | 0.4 | 86.2 | 8.4 | 0.3 | 3.8 | 0.1 | 0.7 | 2 446 | 0.0 | 95.1 | 3.2 | 0.0 | 1.4 | 0.0 | 0.4 | 1 102 |
| Mexico (MEX) | 1998–2003 | 0.9 | 5.3 | 68.8 | 0.5 | 20.5 | 0.7 | 3.3 | 18 283 | 6.9 | 21.5 | 51.3 | 0.7 | 13.4 | 1.5 | 4.7 | 3 590 |
| Nicaragua (NIC) | 1997–2003 | 1.8 | 61.4 | 25.7 | 0.6 | 7.4 | 0.1 | 2.9 | 1 647 | 7.7 | 84.6 | 4.8 | 0.1 | 1.7 | 0.0 | 1.1 | 726 |
| Panama (PA) | 1998–2003 | 1.4 | 18.3 | 63.5 | 0.0 | 11.9 | 3.2 | 1.8 | 816 | 2.9 | 46.3 | 44.1 | 0.0 | 2.2 | 3.7 | 0.7 | 136 |
| Paraguay (PY) | 1996–2003 | 0.6 | 15.4 | 42.9 | 1.6 | 30.4 | 1.2 | 7.9 | 687 | 2.5 | 38.5 | 27.1 | 1.9 | 21.5 | 2.8 | 5.7 | 317 |
| Peru (PE) | 1999–2000 | 2.3 | 54.6 | 14.1 | 3.3 | 11.8 | 0.3 | 13.5 | 304 | 1.8 | 83.0 | 7.3 | 2.4 | 1.2 | 0.0 | 4.2 | 165 |
| Puerto Rico (PR) | 1999–2002 | 4.8 | 1.6 | 67.6 | 1.7 | 17.6 | 1.5 | 5.1 | 993 | 19.2 | 6.2 | 42.3 | 1.5 | 6.9 | 7.7 | 16.2 | 130 |
| United States of America (USA) | 1999–2002 | 7.1 | 0.3 | 20.4 | 0.9 | 60.6 | 1.9 | 8.8 | 97 014 | 31.0 | 0.5 | 16.9 | 2.1 | 35.7 | 3.4 | 10.5 | 23 629 |
| Uruguay (ROU) | 1997–2001 | 1.5 | 1.5 | 41.1 | 2.7 | 47.8 | 1.1 | 4.2 | 2 027 | 6.8 | 3.7 | 27.5 | 9.1 | 35.7 | 7.6 | 9.5 | 484 |
| Venezuela (YV) | 1996–2002 | 1.4 | 13.3 | 56.6 | 0.6 | 23.3 | 2.2 | 2.6 | 7 021 | 5.2 | 29.8 | 44.1 | 0.5 | 12.2 | 4.6 | 3.7 | 1 395 |

(*Continued*)

**Table 3.5 (*Continued*)** **Suicide by method (% out of all suicides by country) according to the WHO Mortality Database (as of November 2006) [36]; countries reporting *ICD-10* data [37]**

| | | Men | | | | | | | | Women | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Country** | **Years** | **Other poisoning** | **Pesticides** | **Hanging** | **Drowning** | **Firearms** | **Falls** | **Other** | ***N*** | **Other poisoning** | **Pesticides** | **Hanging** | **Drowning** | **Firearms** | **Falls** | **Other** | ***N*** |
| **Asia** | | | | | | | | | | | | | | | | | |
| Hong Kong Special Administrative Region, China (HK) | 2001–2004 | 1.6 | 1.1 | 22.6 | 2.0 | 0.3 | 43.3 | 29.1 | 2 866 | 3.5 | 2.4 | 18.9 | 4.5 | 0.1 | 47.5 | 23.1 | 1 556 |
| Israel (IL) | 1998–2003 | 2.5 | 1.9 | 42.0 | 0.7 | 25.4 | 10.3 | 17.2 | 1 511 | 8.9 | 2.9 | 31.1 | 2.1 | 9.1 | 21.9 | 24.0 | 383 |
| Japan (J) | 1995–2004 | 1.3 | 2.5 | 68.7 | 2.6 | 0.2 | 8.1 | 16.5 | 199 505 | 2.9 | 4.3 | 59.9 | 7.8 | 0.0 | 12.5 | 12.7 | 82 646 |
| Kuwait (KWT) | 1995–2001 | 0.5 | 4.7 | 91.7 | 0.0 | 0.5 | 0.5 | 2.1 | 193 | 0.0 | 7.3 | 90.6 | 0.0 | 0.0 | 2.1 | 0.0 | 96 |
| Republic of Korea (ROK) | 1995–2004 | 0.4 | 37.5 | 39.2 | 3.2 | 0.4 | 9.5 | 9.8 | 53 449 | 0.8 | 42.8 | 26.0 | 3.8 | 0.1 | 18.5 | 8.1 | 23 392 |
| Thailand (T) | 1994–2002 | 6.3 | 16.4 | 51.7 | 0.1 | 6.1 | 0.1 | 19.3 | 27 015 | 11.3 | 28.3 | 41.8 | 0.1 | 1.9 | 0.2 | 16.4 | 8 669 |
| **Australia & New Zealand** | | | | | | | | | | | | | | | | | |
| Australia (AUS) | 1998–2003 | 8.0 | 1.1 | 45.4 | 1.3 | 11.5 | 3.6 | 29.1 | 11 422 | 26.5 | 0.7 | 36.4 | 3.9 | 2.6 | 4.6 | 25.3 | 3 017 |
| New Zealand (NZ) | 2000–2003 | 6.4 | 1.0 | 48.4 | 1.9 | 11.2 | 2.5 | 28.6 | 1 493 | 19.7 | 0.4 | 42.5 | 4.4 | 2.2 | 6.4 | 24.3 | 456 |
| **Europe** | | | | | | | | | | | | | | | | | |
| Austria (A) | 2002–2005 | 5.6 | 0.3 | 48.1 | 3.3 | 20.7 | 8.9 | 13.1 | 4 373 | 17.7 | 0.6 | 35.2 | 10.7 | 2.6 | 18.1 | 15.1 | 1 444 |
| Croatia (HR) | 1995–2004 | 2.3 | 1.5 | 53.3 | 3.8 | 25.4 | 4.0 | 9.8 | 6 892 | 7.2 | 5.4 | 47.9 | 13.8 | 4.5 | 8.3 | 12.9 | 2 426 |
| Czech Republic (CZ) | 1994–2004 | 5.0 | 0.6 | 63.8 | 1.0 | 12.4 | 6.5 | 10.8 | 14 154 | 18.2 | 1.3 | 44.8 | 4.8 | 2.6 | 15.7 | 12.5 | 4 016 |
| Denmark (DK) | 1994–2001 | 13.7 | 0.7 | 40.7 | 4.8 | 14.5 | 5.1 | 20.5 | 4 645 | 36.9 | 0.7 | 29.6 | 13.2 | 0.8 | 7.9 | 10.7 | 1961 |
| Estonia (EST) | 1997–2005 | 1.5 | 0.2 | 79.7 | 0.5 | 9.1 | 3.3 | 5.7 | 2 874 | 9.1 | 1.9 | 70.4 | 2.2 | 1.3 | 10.7 | 4.3 | 689 |
| Finland (FIN) | 1996–2004 | 17.6 | 0.2 | 33.1 | 3.5 | 26.7 | 4.2 | 14.6 | 8 168 | 49.5 | 0.2 | 20.3 | 10.6 | 2.6 | 6.6 | 10.2 | 2 425 |
| France (F) | 2000–2003 | 8.6 | 1.0 | 48.9 | 3.9 | 22.1 | 4.9 | 10.6 | 31 378 | 26.3 | 2.0 | 29.2 | 12.4 | 4.1 | 12.4 | 13.5 | 11 387 |
| Georgia (GE) | 1998–2001 | 4.3 | 3.6 | 53.2 | 0.9 | 3.2 | 1.6 | 33.2 | 440 | 4.7 | 3.9 | 50.8 | 0.8 | 0.8 | 4.7 | 34.4 | 128 |
| Germany (D) | 1998–2004 | 8.0 | 1.3 | 55.5 | 2.1 | 10.3 | 7.4 | 15.5 | 57 202 | 22.0 | 2.0 | 38.9 | 7.2 | 1.4 | 14.1 | 14.4 | 20 870 |

(*Continued*)

**Table 3.5 (*Continued*)** Suicide by method (% out of all suicides by country) according to the WHO Mortality Database (as of November 2006) [36]; countries reporting *ICD-10* data [37]

| | | Men | | | | | | | | Women | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Country | Years | Other poisoning | Pesticides | Hanging | Drowning | Firearms | Falls | Other | *N* | Other poisoning | Pesticides | Hanging | Drowning | Firearms | Falls | Other | *N* |
| Hungary (H) | 1996–2003 | 7.0 | 4.6 | 70.3 | 1.4 | 4.0 | 4.9 | 7.8 | 19 030 | 28.1 | 7.0 | 43.4 | 4.5 | 0.6 | 9.9 | 6.5 | 6 089 |
| Iceland (IS) | 1997–2004 | 9.3 | 0.0 | 39.0 | 5.5 | 19.5 | 5.1 | 21.6 | 236 | 31.8 | 1.5 | 27.3 | 18.2 | 0.0 | 4.5 | 16.7 | 66 |
| Latvia (LV) | 1996–2004 | 0.9 | 1.0 | 85.1 | 0.6 | 6.5 | 2.3 | 3.6 | 5 367 | 6.2 | 4.1 | 72.6 | 3.9 | 0.9 | 7.8 | 4.5 | 1 359 |
| Lithuania (LT) | 1998–2004 | 1.1 | 0.4 | 91.7 | 0.3 | 2.7 | 1.3 | 2.4 | 8 778 | 6.3 | 1.6 | 83.1 | 2.2 | 0.3 | 4.4 | 2.1 | 1 881 |
| Luxembourg (L) | 1998–2004 | 8.1 | 1.1 | 38.2 | 2.8 | 14.6 | 18.5 | 16.6 | 356 | 29.6 | 1.6 | 15.2 | 7.2 | 3.2 | 28.8 | 14.4 | 125 |
| Malta (M) | 1995–2004 | 6.8 | 1.4 | 41.8 | 4.1 | 15.8 | 21.9 | 8.2 | 146 | 13.7 | 2.0 | 15.7 | 7.8 | 0.0 | 56.9 | 3.9 | 51 |
| Moldova (MD) | 1996–2004 | 0.9 | 7.0 | 80.3 | 2.0 | 2.4 | 2.6 | 4.8 | 4 596 | 5.0 | 18.0 | 55.7 | 9.1 | 0.5 | 5.4 | 6.2 | 933 |
| Netherlands (NL) | 1996–2004 | 11.7 | 1.4 | 47.9 | 6.6 | 4.4 | 7.8 | 20.2 | 9 211 | 24.0 | 1.8 | 33.6 | 11.0 | 0.6 | 10.7 | 18.3 | 4 526 |
| Norway (N) | 1996–2004 | 11.1 | 0.2 | 37.9 | 4.6 | 27.1 | 4.7 | 14.3 | 3 519 | 33.3 | 0.5 | 32.3 | 13.5 | 2.0 | 7.1 | 11.3 | 1 272 |
| Poland (PL) | 1999–2004 | 1.8 | 0.3 | 91.2 | 0.5 | 1.1 | 2.1 | 3.1 | 29 808 | 7.9 | 0.8 | 77.6 | 3.0 | 0.2 | 6.5 | 4.0 | 5 495 |
| Portugal (P) | 2002–2003 | 2.4 | 14.0 | 52.2 | 4.3 | 11.1 | 6.0 | 10.0 | 1 835 | 9.2 | 23.5 | 31.2 | 11.6 | 3.2 | 10.3 | 10.9 | 532 |
| Romania (RO) | 1999–2004 | 3.0 | 3.1 | 87.3 | 0.3 | 1.0 | 1.4 | 3.8 | 14 039 | 7.9 | 9.1 | 74.1 | 1.1 | 0.1 | 2.4 | 5.2 | 2 934 |
| Serbia (SRB) | 1997–2002 | 1.6 | 2.9 | 57.6 | 3.3 | 20.1 | 2.3 | 12.2 | 6 939 | 4.2 | 9.8 | 57.2 | 7.9 | 5.2 | 4.0 | 11.7 | 3 003 |
| Slovakia (SK) | 1994–2002 | 3.2 | 1.7 | 70.0 | 0.8 | 12.3 | 5.0 | 7.0 | 5 248 | 17.0 | 2.8 | 50.2 | 2.8 | 2.9 | 17.2 | 7.0 | 983 |
| Slovenia (SLO) | 1997–2004 | 2.5 | 1.8 | 64.7 | 2.5 | 11.8 | 3.6 | 13.1 | 3 538 | 8.6 | 3.1 | 53.1 | 12.2 | 1.2 | 9.0 | 12.8 | 1 040 |
| Spain (E) | 1999–2004 | 3.5 | 2.6 | 52.7 | 3.9 | 7.1 | 18.4 | 11.8 | 15 269 | 8.3 | 5.4 | 29.4 | 7.6 | *0.9* | *36.9* | *11.5* | *4 887* |
| Sweden (S) | 1997–2002 | 16.0 | 0.3 | 39.4 | 5.3 | 17.1 | 4.4 | 17.6 | 5 094 | 42.9 | 0.1 | 25.12 | 12.4 | 0.9 | 7.2 | 11.3 | 2 060 |
| Switzerland (CH) | 2000–2004 | 13.3 | 0.6 | 27.3 | 3.0 | 33.5 | 9.2 | 13.2 | 4 635 | 37.8 | 0.7 | 19.1 | 10.1 | 3.4 | 14.7 | 13.9 | 2 111 |
| United Kingdom (GB) | 2001–2004 | 14.7 | 0.4 | 55.2 | 2.4 | 3.5 | 2.9 | 20.8 | 12 573 | 41.1 | 0.3 | 35.9 | 4.7 | 0.6 | 3.7 | 13.9 | 3 832 |

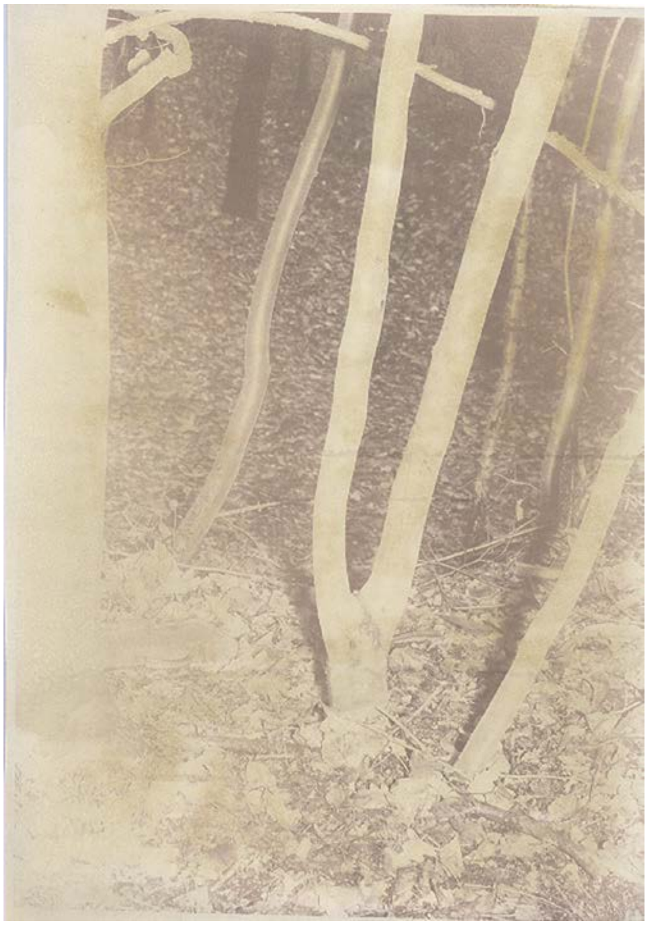


**Figure 3.5** Fork of two branches of a tree.

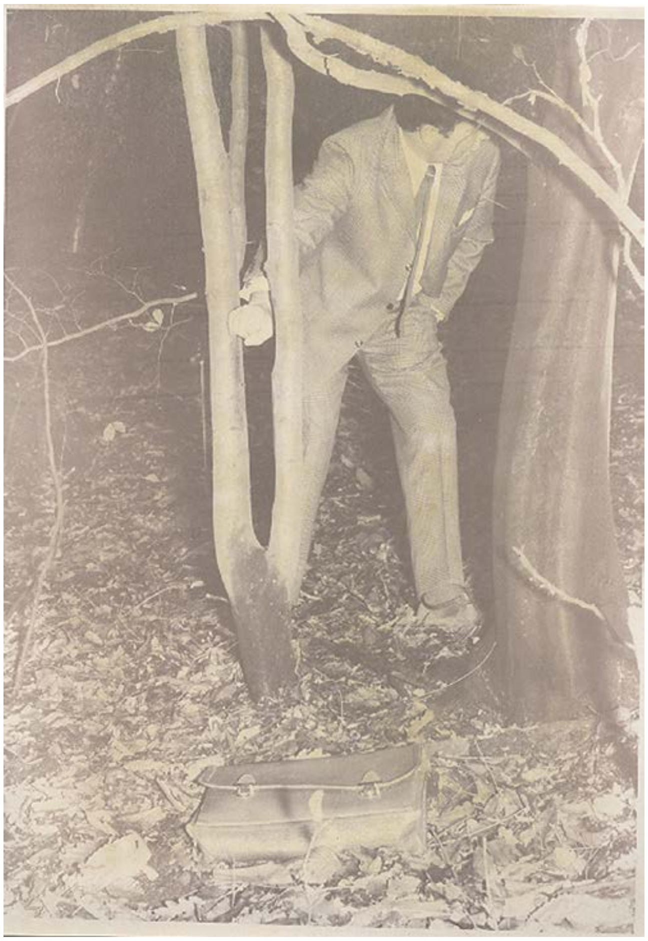


**Figure 3.6** A police officer demonstrating the height at which the neck was fixed in the fork.

death worldwide. By their nature, suicides are obviously underrepresented in official statistics. Three-quarters of suicides globally occur in the developing world. Rates of completed suicides are generally higher in men than in women. Suicide is generally most common among those over the age of 70. However, those aged between 15 and 30 years are at a high risk in certain countries. The most commonly used method of suicide varies between countries. Common methods include hanging, pesticide poisoning and use of firearms. A review of 56 countries found that hanging was the most common method in most of those countries, accounting for 53 per cent of male suicides and 39 per cent of female suicides (Table 3.5).

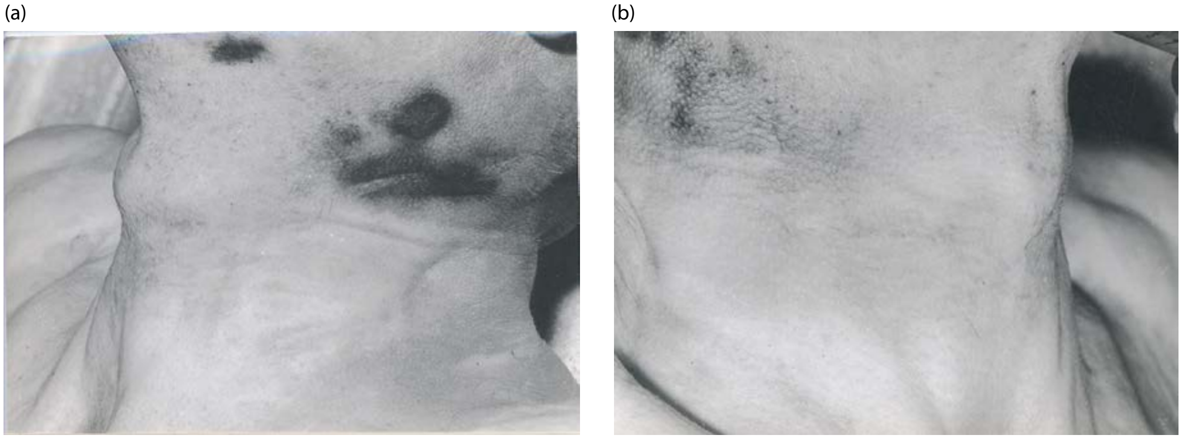


**Figure 3.7** (a), (b) Abrasions on the left and right side of the neck.

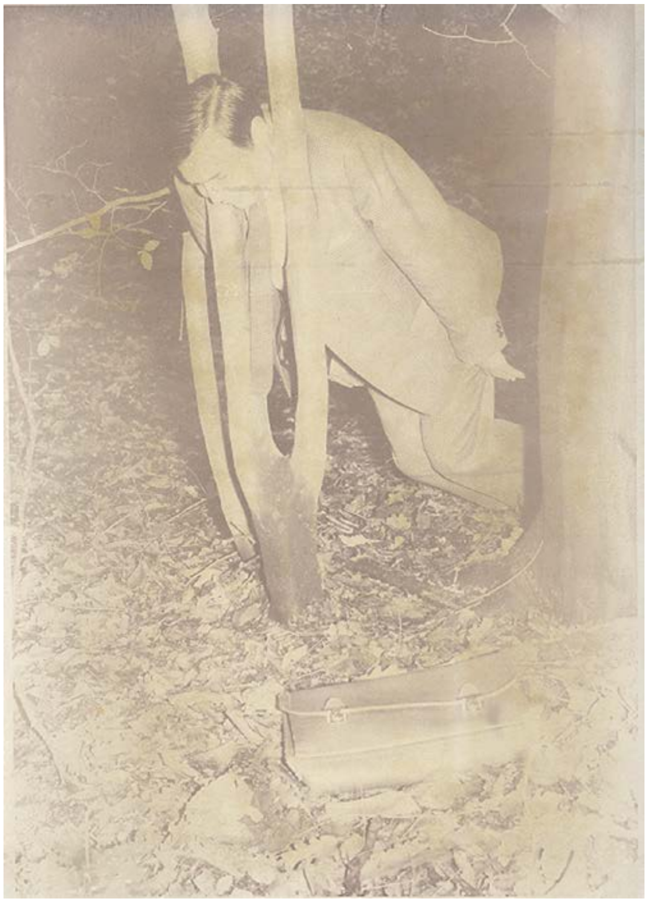


**Figure 3.8** A police officer demonstrating the finding situation.

An *accident* (also known as an *unintentional injury*) is an undesirable, incidental and unplanned event that could have been prevented had circumstances leading up to the accident been recognized, and acted upon, prior to its occurrence.

Accidental asphyxiations are comparatively rare. Accidental carbon monoxide poisoning by charcoal grills is one example. Accidental strangulations in adults are particularly rare. Victims of accidental strangulation are normally either babies/infants, or men who died during an autoerotic act. Accidental strangulation in adulthood not involving an autoerotic act has been described only rarely [4,5,16–18,24,27].

The author has personal experience of one example, a 49-year-old physician who was found in the woods below his clinic shortly after he had been seen alive in the clinic [24]. He was found with his neck in the fork of two branches of a tree (Figure 3.5). After admission to hospital, he was declared dead. Due to the small distance between the branches, the head had not sunk to the base of the fork (Figure 3.6). The victim had obviously tried to take a shortcut and did not use the normal path but walked along a steeply inclined path and accidentally stumbled and fell into the fork of the branches. At autopsy, only abrasions of the right and left side of the neck were found. There were no suicidal tendencies. The circumstances at the scene, the case history and the autopsy findings were indicative of an accident (Figures 3.7–3.9).

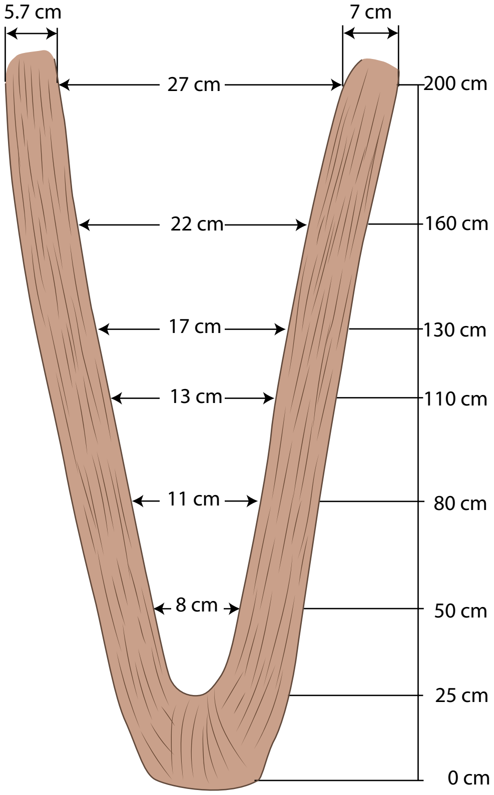


**Figure 3.9** Schematic drawing of the fork indicating the distance between the two branches at different heights.

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# 4 Homicide Methods over Time

*Burkhard Madea*

Homicide methods vary from country to country and have done so over time. In the 19th century, homicide by strangulation and drowning seems to have been very rare in France (Table 4.1).

More than 60 years ago in the United States shooting was the most prevalent homicide method while strangulation was rare (Table 4.2).

In older homicide statistics in different countries the rates of strangulation differ between 3 per cent and 15.4 per cent (Table 4.3). Over the second half of the 20th century, homicide by strangulation became more frequent (17–20%) (Tables 4.4 and 4.5).

If both completed and attempted homicides are taken into account, strangulation is one of the most frequent homicide methods (Table 4.6). In Table 4.7 homicide methods and legal category of homicide are compared.

**Table 4.2 Commitment of 53 homicides in New Hampshire, US (according to Dotzauer et al. [1])**

| Homicide method | Percentage(%) |
|---|---|
| Shooting | 32.1 |
| Blunt force with fist (boxing) | 22.6 |
| Blunt force with instrument | 18.9 |
| Stabbing | 13.2 |
| Strangulation | 1.9 |
| Others | 11.3 |

**Table 4.1 Commitment of homicide in France (1826–1831) (according to Dotzauer et al. [1])**

| | Year | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **Homicide method** | **1826** | **1827** | **1828** | **1829** | **1830** | **1831** | **Sum** | **Percentage (%)** |
| Shooting | 56 | 64 | 60 | 61 | 57 | 88 | **386** | 27.5 |
| Stabbing | 54 | 47 | 42 | 53 | 56 | 64 | **316** | 22.5 |
| Blunt force | 78 | 88 | 94 | 90 | 69 | 79 | **498** | 35.4 |
| Strangulation | 2 | 5 | 2 | 2 | 2 | 4 | **17** | 1.2 |
| Drowning | 6 | 16 | 6 | 1 | 4 | 3 | **36** | 2.6 |
| Kicking | 28 | 12 | 21 | 23 | 17 | 26 | **127** | 9.0 |
| Fire | – | 1 | – | 1 | – | – | **2** | 0.1 |
| Unknown | 17 | 1 | 2 | – | 2 | 2 | **24** | 1.7 |
| **Sum** | **241** | **234** | **227** | **231** | **207** | **266** | **1406** | **100.0** |

**Table 4.3 Homicide methods in different countries* (according to Dotzauer et al. [1])**

| Homicide method | FRENKEL (1930) Ukraine 1920–1929 | UNRUH (1965) Germany 1928–1933 | LODUCHOWSKI (1941) Koblenz 1910–1939 | JACOBS (1952) FRG 1947–1950 |
|---|---|---|---|---|
| Blunt force | 45** (20.8%) | 76 (23.9%) | 10 (15.2%) | 112 (28.3%) |
| Shooting | 76 (35.2%) | 62 (19.5%) | 18 (27.3%) | 134 (33.8%) |
| Strangulation | 31 (14.4%) | 49 (15.4%) | 2 (3.0%) | 59 (14.9%) |
| Stabbing including throat cut | 46*** (21.3%) | 35 (11.0%) | 7 (10.6%) | 44 (11.1%) |
| Poisoning | 3 (1.4%) | 33 (10.4%) | 12 (18.2%) | 12 (3.0%) |
| Drowning | – | 13 (4.1%) | 1 (1.5%) | 4 (1.0%) |
| Fall from height | – | 1 (0.3%) | 1 (1.5%) | – |
| Burning | – | 1 (0.3%) | – | – |
| Kicking | – | 1 (0,3%) | – | – |

*(Continued)*

**Table 4.3 (*Continued*)** Homicide methods in different countries* (according to Dotzauer et al. [1])

| Homicide method | FRENKEL (1930) Ukraine 1920–1929 | UNRUH (1965) Germany 1928–1933 | LODUCHOWSKI (1941) Koblenz 1910–1939 | JACOBS (1952) FRG 1947–1950 |
|---|---|---|---|---|
| Starvation | – | – | 2 (3.0%) | – |
| Other | – | – | 9 (13.6%) | – |
| Unknown | 15 (6.9%) | – | 4 (6.1%) | 31 (7.8%) |
| Combinations | – | 47 (14.8%) | – | – |
| **Sum** | **216 (100%)** | **318 (100%)** | **66 (100%)** | **396 (100%)** |

[*] Cited from UNRUH, FRENKEL, JACOBS, number of perpetrators, LODUCHOWSKI, UNRUH, number of victims;

[**] blunt tool;

[***] sharp tool.

**Table 4.4** Homicides in England and Wales (1957–1968) (according to Dotzauer et al. [1])

| Homicide method | ‘Normal’[*] perpetrator(%) | Victim of ‘abnormal’ perpetrator(%) |
|---|---|---|
| Sharp force injury | 38.7 | 18.9 |
| Blunt force injury | 16.4 | 14.0 |
| Strangulation | 20.8 | – |
| Poisoning | – | 19.1 |
| Stabbing | – | 5.6 |
| Shooting | 9.3 | 13.9 |
| Other | 14.8 | 28.6 |
| **Sum** | **100 (= 573 perpetrators)** | **100 (= 1.257 victims)** |

[*] Male perpetrator, convicted for ‘capital’ and ‘non-capital’ murder.

**Table 4.5** Commitment of homicide in Cologne and Hamburg (1946–1967) (according to Dotzauer et al. [1])

| Homicide method | Percentage (%) |
|---|---|
| Blunt force: | |
| • with instruments | 11.0 |
| • with boxing and kicking | 8.6 |
| Sharp force injury | 17.7 |
| Asphyxiation | 17.2 |
| Poisoning | 15.0 |
| Shooting | 13.9 |
| Run over by car | 3.2 |
| Drowning | 1.7 |
| Other physical causes | 1.4 |
| Fall from height | 1.4 |
| Sum | 91.1 |
| Combination | 8.9 |
| Sum | 100 |

**Table 4.6** Attempted and completed homicide in Germany (according to Dotzauer et al. [1])

| Homicide method | BRÜCKNER (1961) Germany (1942–1956) Homicide | STEIGLEDER (1968) Kiel (1950–1963) Attempted and completed homicide or manslaughter |
|---|---|---|
| Blunt force | 16 (17.6%) | 26 (27.7%) |
| Blunt force and others | 23 (25.3%) | – |
| Shooting | 13 (14.3%) | 15 (16.0%) |
| Shooting and others | 2 (2.2%) | – |
| Stabbing | 10 (11.0%) | 14 (14.9%) |
| Stabbing and others | 3 (3.3%) | – |
| Strangulation | 9 (9.9%) | 22 (23.4%) |
| Strangulation and others | 5 (5.5%) | – |
| Poisoning | 3 (3.3%) | 15 (16,0%) |
| Others | 7 (7.7%) | 2 (2.1%) |
| **Sum** | **91 (100%)** | **94 (100%)** |

**Table 4.7** Mode of commitment of homicides ($n = 195$) between 1989 and 1999 (Autopsies carried out in the Institute of Forensic Medicine, Bonn University [2])

| Mode of commitment | Murder | Manslaughter | Bodily harm with fatal consequences | 'Homicide'* | Sum |
|---|---|---|---|---|---|
| Stabbing | 22 | 26 | 4 | 3 | **55** |
| Blunt force | 19 | 5 | 18 | – | **42** |
| Shooting | 20 | 10 | – | 2 | **32** |
| Ligature strangulation | 7 | 7 | – | 1 | **15** |
| Throttling | 2 | 8 | – | 1 | **11** |
| Obstruction of respiratory orifices | 1 | 2 | – | – | **3** |
| Drowning | 1 | – | – | – | **1** |
| Fire | – | – | 1 | 2 | **3** |
| Combinations | 14 | 9 | 2 | 1 | **26** |
| Other mode of commitment | 1 | 1 | 3 | 2 | **7** |
| **Sum** | **87** | **68** | **28** | **12** | **195** |

* 'Homicide': judicial not (yet) qualified.

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# 5 Case Series on Homicidal Strangulation
## *Criminalistic and Forensic Pathology*

*Burkhard Madea*

According to DiMaio [2], homicides due to asphyxia are relatively uncommon. However, this may be due to the fact that, in the United States, civilian handgun ownership is high.

DiMaio reviewed the files of the Bexar County Medical Examiner's Office from 1 January 1985 to 31 December 1998 for all such homicides. Altogether, 133 cases were found. The largest category was ligature strangulation with 48 deaths (21 male, 27 female) (Tables 5.1 and 5.2).

Petechiae were present in the conjunctiva and/or sclera in 86 per cent of the cases (Table 5.3). Fractures of the hyoid and/or thyroid cartilage were present in 12.5 per cent.

There were a total of 41 deaths from manual strangulation (27 female, 14 male). Motives in female cases of manual strangulation were mainly rape and domestic disputes (Table 5.4).

Petechiae were present in 89 per cent of the cases (Table 5.5). In cases of manual strangulation, fractures of the hyoid, thyroid or cricoid cartilage were found in all the male victims and slightly more than half of the female victims (Table 5.5). The distribution of fractures is shown in Table 5.6. Twenty-six cases of suffocation were found; 20 of the victims were under 2 years of age (Table 5.7). Only one of these children had petechiae and/or scleral haemorrhage.

**Table 5.1 Asphyxial homicides**

| Method | Number of cases |
|---|---|
| Ligature strangulation | 48 |
| Manual strangulation | 41 |
| Suffocation | 26 |
| Choking | 5 |
| Combined methods | 9 |
| Drowning | 3 |
| Hanging | 1 |
| **Total** | **133** |

*Source:* From DiMaio VJM. *Am J Forensic Med Pathol* 2000;**21**(1):1–4 with permission of Wolters Kluwer Health, Inc.

**Table 5.2 Ligature strangulations: motives**

| Motive | Number of victims Male | Number of victims Female |
|---|---|---|
| Rape | 0 | 18 |
| Homosexual dispute | 5 | 0 |
| Burglary/robbery | 3 | 2 |
| Domestic dispute | 0 | 3 |
| Personal dispute | 3 | 0 |
| Drugs- or gang-related | 2 | 2 |
| Unknown | 8 | 2 |
| **Total** | **21** | **27** |

*Source:* From DiMaio VJM. *Am J Forensic Med Pathol* 2000;**21**(1):1–4 with permission of Wolters Kluwer Health, Inc.

**Table 5.3 Homicidal asphyxia**

| Signs | Number of cases evaluated | Percent | |
|---|---|---|---|
| Petechiae | 36 | 31 | (86%) |
| Fractures: | | | |
| Male victims | 21 | 5 | (23.8%) |
| Female victims | 27 | 1 | (3.7%) |
| Total | 48 | 6 | (12.5%) |

*Source:* From DiMaio VJM. *Am J Forensic Med Pathol* 2000;**21**(1):1–4 with permission of Wolters Kluwer Health, Inc.

**Table 5.4 Manual strangulation: motives**

| Motive | Number of victims Male | Number of victims Female |
|---|---|---|
| Rape | 0 | 14 |
| Homosexual dispute | 0 | 10 |
| Burglary/robbery | 1 | 0 |
| Domestic dispute | 5 | 1 |
| Personal dispute | 2 | 0 |
| Drugs- or gang-related | 1 | 0 |
| Unknown | 5 | 2 |
| **Total** | **14** | **27** |

*Source:* From DiMaio VJM. *Am J Forensic Med Pathol* 2000;**21**(1):1–4 with permission of Wolters Kluwer Health, Inc.

**Table 5.5 Manual strangulation: petechiae and fractures of the hyoid, thyroid and cricoid cartilage**

| Signs | Number of cases evaluated | Percent | |
|---|---|---|---|
| Petechiae | 37 | 33 | (89%) |
| Fractures | | | |
| Male victims | 14 | 14 | (100%) |
| Female victims | 27 | 14 | (52%) |
| Total | 41 | 28 | (68.2%) |

*Source:* From DiMaio VJM. *Am J Forensic Med Pathol* 2000;**21**(1):1–4 with permission of Wolters Kluwer Health, Inc.

**Table 5.6 Manual strangulation: distribution of fractures**

| Structure | Number of victims<br>Male | Female | Total |
|---|---|---|---|
| Hyoid only | 4 | 5 | **9** |
| Thyroid only | 2 | 0 | **2** |
| Cricoid only | 2 | 0 | **2** |
| Hyoid–thyroid | 5 | 4 | **9** |
| Hyoid–cricoid | 0 | 3 | **3** |
| Hyoid–thyroid–cricoid | 1 | 2 | **3** |
| **Total** | **14** | **14** | **28** |

*Source:* From DiMaio VJM. *Am J Forensic Med Pathol* 2000;**21**(1):1–4 with permission of Wolters Kluwer Health, Inc.

**Table 5.7 Petechiae in cases of suffocation**

| Victim | Number of cases evaluated | Present |
|---|---|---|
| Child ≤2 years of age | 18 | 1 |
| Adult | 6 | 2 |

*Source:* From DiMaio VJM. *Am J Forensic Med Pathol* 2000;**21**(1):1–4 with permission of Wolters Kluwer Health, Inc.

**Table 5.9 Homicide methods**

| Method | Copenhagen* | | | Oslo* | | |
|---|---|---|---|---|---|---|
| | Male | Female | Total | Male | Female | Total |
| Blunt force | 10.2 | 5.8 | **16.0** | 16.7 | 4.5 | **21.2** |
| Sharp force | 22.9 | 13.1 | **36.0** | 18.5 | 8.4 | **26.9** |
| Strangulation | 4.4 | 18.5 | **22.9** | 8.3 | 12.2 | **20.5** |
| Gunshot | 10.9 | 7.3 | **18.2** | 11.5 | 9.6 | **21.1** |
| Combination** | 1.5 | 2.5 | **4.0** | 3.2 | 3.2 | **6.4** |
| Arson | 0.7 | 0 | **0.7** | 1.3 | 1.3 | **2.6** |
| Others | 0.4 | 0.7 | **1.1** | 0.6 | 0.6 | **1.2** |
| Unknown | 0.7 | 0.4 | **1.1** | 0 | 0 | **0** |

*Source:* From Hougen HP, Rodge S, Poulsen K. *Am J Forensic Med Pathol* 1999;**20**(2):293–299 with permission of Wolter Kluwers, Inc.

* Each number indicates percentage of total number of victims in each city (Copenhagen = 274 victims; Oslo = 156 victims).

** Combination of two or more methods.

Five deaths were due to choking. Three of the deaths involved adults who were gagged; two deaths involved infants with foreign material pushed into the mouth. Other categories of asphyxia were as follows: Nine deaths were due to more than one form of asphyxia (Table 5.8); one death was due to hanging; and three deaths were due to drowning.

Rape was the motive in 66 per cent of the female victims of ligature strangulation and 52 per cent of those due to manual strangulation.

Hougen et al. [3] investigated homicides in the two Scandinavian capitals Copenhagen (Denmark) and Oslo (Norway), for the 10-year period from 1985 to 1994. The total number of homicides was 431; 63.8 per cent occurred in Copenhagen and 36.2 per cent in Oslo. The average homicide rate was 1.6 per 100,000 in Copenhagen and 1.8 per 100,000 in Oslo. Blunt force, sharp force and strangulation were the most common methods (Table 5.9). In females, in both Copenhagen and Oslo, strangulation was the most prevalent homicide method. The clear female preponderance in both cities was more marked in Copenhagen than in Oslo. There was an increase in homicides over weekends. Most perpetrators were known to their victims.

Further analyses of the cases from Oslo and Copenhagen revealed that 95 asphyxial homicides were committed in the 10-year period 1985–1994, accounting for 22 per cent of all homicides in that period [5]. Sixty-nine (73%) of the asphyxia victims were female (Figure 5.1). The most common method of asphyxiation was manual strangulation (Figure 5.2). Seventeen (18%) of the victims were under the age of 10 years, accounting for 59 per cent of all homicides

**Table 5.8 Deaths due to more than one form of asphyxia**

| Form of asphyxia | Number of cases |
|---|---|
| Manual/ligature strangulation | 7 |
| Manual strangulation/suffocation | 1 |
| Ligature strangulation/choking (gag) | 1 |

*Source:* From DiMaio VJM. *Am J Forensic Med Pathol* 2000;**21**(1):1–4 with permission of Wolters Kluwer Health, Inc.

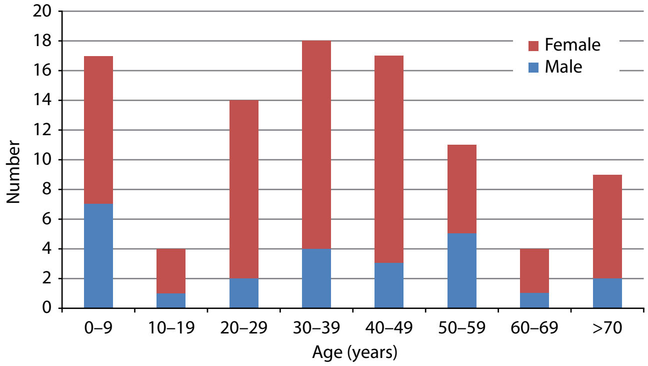

**Figure 5.1** Age and gender of the asphyxia homicide victim population. The number of victims is given on the *y*-axis, age intervals (in years) on the *x*-axis (from Rogde S, Hougen HP, Poulsen K. *Am J Forensic Med Pathol* 2001;**22**(2):128–133 with permission from Wolters Kluwer Health, Inc.).

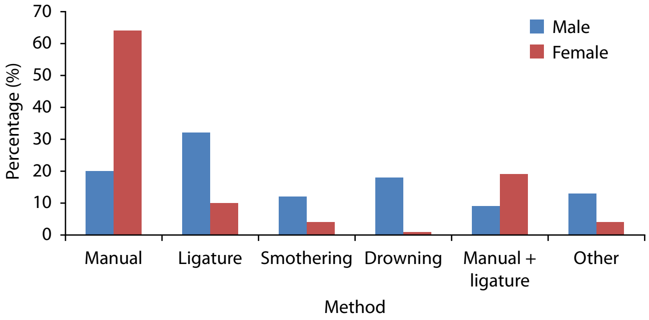

**Figure 5.2** Asphyxia method/weapon. The percentage of victims of each gender is indicated on the *y*-axis, the various methods are given on the *x*-axis (from Rogde S, Hougen HP, Poulsen K. *Am J Forensic Med Pathol* 2001;**22**(2):128–133 with permission from Wolters Kluwer Health, Inc.).

in that age group. Whereas 38 per cent of female victims were killed by their spouse, this was the case for only one male victim. The motive was not known in a great proportion of cases. Fifty-six per cent of victims had been subjected to additional violence and, in this respect, there was no difference between the sexes. In 12 of the cases, the offender was female; in 9 such cases, the victim was her offspring. More than half of the victims had no blood alcohol. The crime scene was the victim's domicile among 72 per cent of the female and 52 per cent of the male victims. Forty-two per cent of the female and 11 per cent of the male victims over the age of 10 years were married or cohabitants. The authors concluded that asphyxia homicides seem to be a method favoured by a physically superior person to a victim with considerably less physical strength. This is illustrated by the greater proportion of female and child victims. Also, most of the female perpetrators in this retrospective study (9 out of 11) murdered their own children.

Brendel [1] reviewed 54 homicides by manual strangulation, ligature strangulation, suffocation and drowning from 1993 to 1999 which were investigated at the Institute of Forensic Medicine, University of Münster. These 54 homicides by 'asphyxia' comprised 90 per cent of all homicides investigated during this period. In 17 cases, cause of death was either manual or ligature strangulation, in 9 persons suffocation, 2 persons were drowned. More than half of the homicides were committed between 10 p.m. and 6 a.m. In 57 per cent the reason for the homicide was intra-familiar struggle, and in 37 per cent the victim and perpetrator were married. More than 70 per cent of the victims were female, the majority between 21 and 30 years of age. Eighty-eight per cent of the perpetrators were male, mostly between 21 and 40 years of age. One-third of the victims were alcoholized at the time of death. Thirty per cent of the perpetrators were unemployed. Nearly one-third of the perpetrators were known to the police, mostly due to alcohol dependence. While committing the homicide, 53 per cent of the perpetrators were alcoholized. In 70 per cent of the intra-familiar homicides, the motive was a dispute or jealousy. In the extra-familiar homicides, motives were sexual or robbery.

A retrospective analysis by Padosch et al. [4] on homicides from 1989 to 1999 revealed 195 cases in total. In 15 cases, the homicide was committed by ligature strangulation, in 11 by manual strangulation, in 3 by suffocation and in 7 by drowning. The typical perpetrator was a male with a mean age of 35 years, driven by personal motives or greed within his immediate vicinity, and the crime affected family members or close acquaintances.

A review of 300 attempted strangulation cases by Strack et al. [6] revealed that, although the victims in the study did not die, they did nevertheless experience a serious and potentially lethal form of asphyxia violence. Understandably, the police and prosecutors focused on visible injuries to prove strangulation; other symptoms were largely overlooked. No one knew what questions to ask the victim to determine if she had any symptoms such as voice changes, breathing changes or pain from swallowing. Eighteen per cent of the strangulation victims reported pain (either to the neck or throat), 1 per cent voice changes, 5 per cent breathing changes and 2 per cent problems with swallowing. The authors concluded that a lack of training may have caused the police and prosecutors to overlook symptoms of strangulation or to rely too heavily on the visible signs of strangulation only. Because most victims of strangulation had no visible injuries or the injuries were too minor to photograph, opportunities for higher level criminal prosecution were missed.

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# 6

# Pathophysiology

*Wolfgang Keil and Claire Delbridge*

## Definition and terms

In forensic medicine, asphyxiation as a cause of death means that brain death occurs immediately as a result of a traumatically caused $O_2$ deficiency [27]. In these cases it is a non-natural death.

Asphyxiation can also occur non-traumatically, i.e. naturally. This applies to all internal diseases in which fatal aspiration of vomit or blood occurs as a complication. These include gastrointestinal (GI) diseases such as ileus and oesophageal variceal bleeding.

The traumatic $O_2$ deficiency of the brain is essentially caused by three factors:

- Respiratory disorders
- Circulatory disturbances
- Increased $O_2$ requirement

In practice, a combination of all three factors is predominant, with each individual disorder having a different significance, depending on the type of asphyxiation and the specific circumstances surrounding the death.

Occasionally, high-grade cerebral $O_2$ deficiency is survived for a longer period. Those affected, who have undergone such near-asphyxiation, always have irreversible cerebral deficits.

It is widely accepted that 'asphyxiation' is a general term for a group of causes of death in which brain death occurs immediately as a result of $O_2$ deficiency [27,55]. Occasionally, the term 'asphyxia' is used synonymously with 'asphyxiation' in forensic pathology. It seems problematic that both terms are sometimes used with different meanings in international literature. Both 'asphyxiation' and 'asphyxia' are often used to refer to special types of lethal $O_2$ deficiency. For example, Eisenmenger and Gilg [15] and Knight [31] use 'asphyxiation' as a synonym for 'suffocation', meaning an obstruction of mouth and nose. DiMaio and DiMaio [12] use the term 'asphyxia' mainly for cases with so-called 'mechanical asphyxia', wanting to characterize compression of the thorax and/or the abdomen with breathing impairment. The heterogeneity of the terminology considerably complicates the presentation and ability to compare the data of different authors. Examples of the different meanings of individual terms in English literature can be found in Table 6.1.

In the absence of binding definitions, one and the same finding can be described as smothering or suffocation, for example.

Thus, it is critical in forensic pathology not only to define and apply the term 'asphyxiation' uniformly, but also to introduce an internationally uniform nomenclature for the individual subtypes of 'asphyxiation'. A future classification of the individual forms of asphyxiation should be based on the concrete ways of development and the anatomical regions concerned, so that the diagnoses are comprehensible for everyone. Terms such as 'suffocation', which require further interpretation, should be avoided. The standardization of terminology not only facilitates the presentation of facts in court but is also the basis for the categorization and comparison of scientific data on asphyxiation collected worldwide.

In this sense, the terminology of the subtypes of 'asphyxiation' compiled in Figure 6.1 was developed. It

**Table 6.1 Terms for undefined subtypes of 'asphyxiation' and their different meanings (examples according to Sauvageau and Boghossian [55])**

| | Meaning(s) | |
|---|---|---|
| **Term** | **Common** | **Less common** |
| Suffocation | $O_2$ deficiency in the breathing air or in closed rooms | Narrowing or closure of the breathing cavities |
| Smothering | Narrowing or closure of outer airway passages | Outer obstruction of upper airways<br>Form of suffocation with compression or closure of mouth and nose |
| Choking | Narrowing or obstruction of the upper airways by foreign bodies (also bolus) | Obstruction of mouth, oropharynx, larynx |
| Entrapment | Suffocation = $O_2$ deficiency in breathing air or enclosed spaces<br>Environmental suffocation | Asphyxia with confined and enclosed spaces |
| Traumatic asphyxiation | Chest compression by a heavy weight | Postural asphyxia and positional asphyxia |

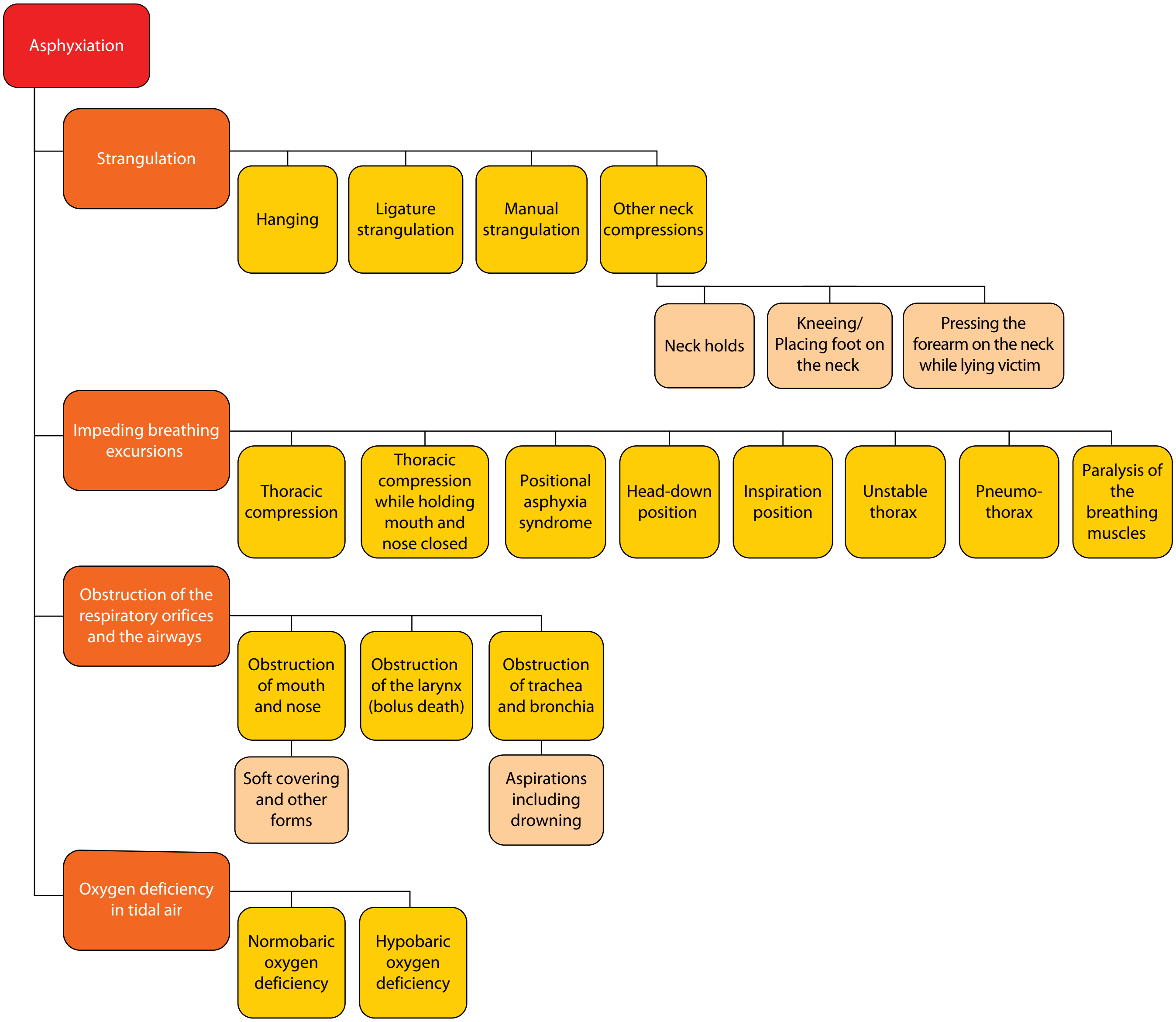
Asphyxiation
Strangulation
Hanging
Ligature strangulation
Manual strangulation
Other neck compressions
Neck holds
Kneeing/ Placing foot on the neck
Pressing the forearm on the neck while lying victim
Impeding breathing excursions
Thoracic compression
Thoracic compression while holding mouth and nose closed
Positional asphyxia syndrome
Head-down position
Inspiration position
Unstable thorax
Pneumo-thorax
Paralysis of the breathing muscles
Obstruction of the respiratory orifices and the airways
Obstruction of mouth and nose
Obstruction of the larynx (bolus death)
Obstruction of trachea and bronchia
Soft covering and other forms
Aspirations including drowning
Oxygen deficiency in tidal air
Normobaric oxygen deficiency
Hypobaric oxygen deficiency

**Figure 6.1** Classification of asphyxiation in forensic pathology (modified image [27]).

partly considers classifications that have already been used internationally [27,55].

The four main groups depicted in Figure 6.1 result from the way various forms of asphyxiation originate. At the same time, the compilation considers the anatomical regions concerned and the pathophysiological effects.

In regard to terminology, it should be remembered that the term 'asphyxia' is often used in clinical medicine. This term comes from Greek and means pulselessness. It is used for patients suffering from respiratory depression with circulatory weakness. Hypoxaemia and hypercapnia are usually present, so that those affected are cyanotic. There are no defined criteria for the use of this term. 'Asphyxia' refers to a complexity of symptoms in patients that can have different causes. Thus, the term is not compatible with the meaning in forensic pathology. The term 'asphyxia' should therefore be avoided in morphology in favour of the term 'asphyxiation'.

It should be noted that internal diseases, injuries and intoxications can lead to acute respiratory insufficiencies with diffusion disorders in the lungs as well as impairments of the transport, binding or release of $O_2$ in the body. Finally, the developing $O_2$ deficiency – in contrast to the traumatic events – indirectly causes hypoxic-ischaemic brain damage. If death occurs as a result of the underlying disease (e.g. due to a myocardial infarction with reduced or interrupted haemoperfusion of the brain), the term 'hypoxic brain death' is used in clinical medicine, not the term 'asphyxiation' [34]. In such cases, it is customary to then state the underlying condition (e.g. acute myocardial infarction, pulmonary embolism or bleeding to death in the GI tract), as the direct cause of death, although death was ultimately caused pathophysiologically by indirect hypoxic-ischaemic brain death. Hypoxia leading to death by CO and $CN^-$ are accordingly referred to as 'intoxications' because indirect hypoxic brain damage is the cause of death.

## $O_2$ deficiency traumas and consequences: Overview

The traumas that lead to cerebral hypoxia can affect the entire organism, affect only individual parts of the body or affect the respiratory organs. The pathomechanisms vary according to the variety of traumatic influences (Table 6.2).

All the impairments listed in Table 6.2, which occur practically in combination, result in an $O_2$ deficit with damage to nerve cells and may result in brain death. Brain death occurs because the brain has by far the greatest $O_2$ sensitivity of all organs. It is known that the normal value of $paO_2$ in humans is 75–97 mmHg. Respiratory insufficiencies are associated with $paO_2$ values of $\leq 60$ mmHg. Acute hypoxemic conditions at $\leq 50$ mmHg are to be regarded as critical. Cerebral hypoxic damage occurs at values of $\leq 36$ mmHg. A $paO_2 < 20$ mmHg is always acutely life-threatening [34,43]. At such drops of $paO_2$ the total $O_2$ concentration of arterial blood ($CaO_2$) decreases from about 20 ml/dl to values below 12 ml/dl. However, these values, which are taken from clinical medicine, have no significance for the postmortem diagnosis of asphyxiation deaths. This is because extremely low $O_2$ concentrations are formed during every process of dying as a result of the failure of vital functions, irrespective of the cause of death. Postmortem, $O_2$ consumption of the tissues can lead to a further decrease in these values.

## $O_2$ deficit in breathing gas: Hypoxia

If the concentration or partial pressure of $O_2$ in the inhaled air, or other inhaled gas is reduced, hypoxaemia, general and cerebral hypoxia occur. Initially, the alveolar gas exchange is not impaired. As a result, $CO_2$, which accumulates under the rising $O_2$ deficit, can be breathed out at the beginning of the asphyxiation process, and the reactions that are normally triggered by increased $CO_2$ levels in the blood are initially absent. Thus, respiratory distress, restlessness and fear of asphyxiation or death are not initially perceived by those affected or are not perceived with significant intensity. Since the unnoticed $O_2$ deficiency usually ultimately leads to sudden unconsciousness, attempts at self-rescue in such cases are hardly observed.

**Table 6.2 Impairment of body regions, organs and other factors that can lead to cerebral hypoxia via various pathophysiological effects**

| Body region, organ, other factors | Impairment | Significant pathophysiological consequences |
|---|---|---|
| Environment, breathing gas | Decrease or complete absence of $O_2$ | $O_2$ partial pressure in alveoli↓ |
| Respiratory orifices/upper respiratory tract | Narrowing or displacement of mouth and/or nose and/or throat | |
| Neck | Obstruction of larynx and/or trachea during aspiration | |
| | Compression of larynx and/or trachea as well as veins and arteries | $O_2$ partial pressure in alveoli↓<br>Cerebral blood flow↓ as a result of direct vascular compression and possibly triggering of the carotid sinus reflex |
| Thorax | Moderate compression with obstruction of respiratory movements | $O_2$ partial pressure in alveoli↓ |
| | Strong compression with cancellation of breathing movements and fixation in expiratory position | $O_2$ partial pressure in alveoli↓<br>Cerebral blood flow↓ as a result of increase in thoracic intracranial pressure |
| | Pneumothorax | |
| Bronchi | Constriction or luminal obstruction | $O_2$ partial pressure in alveoli↓ |
| Lungs | Obstruction of the alveoli due to foreign body or edema | |
| Body position | Especially head-down positions, or obstructions of breathing movements possibly also in prone position | Cerebral blood flow↓ due to congestion of blood in the head and neck area<br>$O_2$ partial pressure in alveoli↓ as a result of obstructions of the respiratory auxiliary musculature and/or the diaphragmatic breathing |
| Psychic/physical excitement | Under stress catecholamine release, especially adrenaline-serum levels↑ | $O_2$ requirement of all organs and tissues, especially the skeletal muscle↑ with the consequence of a cerebral $O_2$ deficit |

There are two distinct forms of hypoxia, i.e. normobaric and hypobaric.

### Normobaric hypoxia

The air pressure remains constant. At sea level it is 760 mmHg (= Torr). However, the normal $O_2$ concentration of around 21 per cent by volume (vol%) and thus also the $O_2$ partial pressure in the air we breathe ($pIO_2$) decreases, because other gases such as $CO_2$ or $N_2$, for example, take up larger volume proportions in the gas mixture. The values of the $O_2$ pressure in the alveolar air ($pAO_2$) and in the arterial capillaries ($paO_2$) decrease proportionally to the reduction of the $pIO_2$. The reduction of the $O_2$ concentration in the inspiratory air from 21 vol% to about 10 vol% leads to acute life-threatening conditions because $paO_2$ values of $\leq$50 mmHg result.

The $O_2$ concentration may be reduced by repeated inhalation of one's own exhalation air if only a limited volume sealed against the normal air is available, for example, in containers or in plastic bags pulled over the head.

In extreme cases, gas is initially inhaled with an $O_2$ concentration that is well below 10 vol%, contains only traces of $O_2$, or even contains none at all.

When assessing normobaric hypoxia, it should always be taken into account that not only the reduction of the $O_2$ content (i.e. asphyxiation) plays a role in the occurrence of death, but also possible toxic properties of the gases released (e.g. $CO_2$ or $H_2S$) can be of significance. $CO_2$ intoxications occur at levels >8 vol% in ambient air, which can cause loss of consciousness, coma and death independently of a decreased $O_2$ concentration. Inhalation of $H_2S$ leads to enzyme inhibition.

### Hypobaric hypoxia

In hypobaric hypoxia, the air pressure is reduced and therefore also the $paO_2$. However, the volume fraction of $O_2$ in the ambient air of around 21 per cent remains unchanged. This constellation requires a high-altitude exposure, because with increasing altitude the air pressure and thus also the $O_2$ partial pressure in the inspiratory air decrease. At a height of 5.5 km, the $pIO_2$ in the atmospheric air is only half the amount found at sea level, so that the $paO_2$ reaches the critical range of $\leq$50 mmHg and cerebral hypoxia can lead to brain death.

## Obstructions of breathing: Hypoventilation

Forensic medicine is primarily concerned with the traumatic causes that can lead to a reduction in normal lung ventilation (i.e. hypoventilation). In such events, normal concentration and pressure conditions exist in the ambient air.

Hypoventilation initially leads to a drop in the $O_2$ content in the blood, and hypoxaemia develops. If at least 30 per cent of the blood is deoxygenated, it turns bluish according to its absorption properties, which is particularly noticeable in venous blood. The discolouration is usually visible on the lips. But the mucous membranes, the nail beds and in pronounced cases even the skin can also show a blue discolouration. The increase in hypoxaemia is accompanied by an increase in $CO_2$ concentration, which is usually 40 mmHg in arterial blood. Hypercapnia is defined as $paCO_2$ values of >45 mmHg. This leads to respiratory acidosis. The increase in $paCO_2$ initiates counter-regulations that are clinically perceptible. Especially in the first phase of these asphyxiation processes, tachycardia, extrasystoles and an increase in blood pressure occur. The facial skin reddens as a result of $CO_2$-induced vasodilation. Higher $paCO_2$ concentrations initially cause central attenuation and then lead to loss of consciousness and coma [29,34]. The increase in $paCO_2$ is also attributed to the occurrence of muscle twitches and seizures. Survivors report panic and fear of death, also associated with elevated $paCO_2$ levels.

The following events are major causes of hypoventilation in forensic medicine:

- Obstructions of respiratory openings and passages.
- Neck and chest compression.
- Impairment of respiratory movements.
- Reduction of gas exchange in the alveoli.

### Obstructions of respiratory openings and passages

This includes partial or complete closure of the mouth and nose, for example by covering with objects or by holding with the hands. The oral cavity and the nasopharynx can be narrowed or closed during the gagging process. The same applies to aspirations of chewed food, blood and other liquids as well as objects into the trachea and/or bronchi.

Isolated closures of the larynx, mostly caused by food ingestion, are generally suitable for acute hypoventilation. In most cases, however, a vagal reflex obviously causes a sudden cardiac arrest. If death occurs due to a reflex, this is referred to as bolus death. Hypoventilation is apparently of subordinate importance for the onset of death, since no corresponding symptoms such as coughing or increased breathing movements can be observed in those affected.

### Neck and chest compression

Breathing is hindered by external pressure on the corresponding regions of the body, resulting in hypoventilation. Isolated compression of the larynx and/or trachea is virtually impossible along the neck. In almost all cases, blood vessels are compressed, more or less intensively, at the same time so that blood circulation in the brain is impaired (see 'Compression of the neck'). The resulting cerebral impairment of the $O_2$ supply is usually of much greater significance for asphyxiation than the simultaneous hypoventilation caused by narrowing of the airways. In the case of neck compressions, an additional drop in blood pressure triggered by the reflective effect can occur, which obviously has additional significance for the asphyxiation process in isolated cases.

In mild to moderate thoracic compressions, hypoventilation is the main symptom due to restricted respiratory movements. When the thorax is compressed to a high degree, hypoventilation is accompanied by a reduction in the return flow of venous blood to the heart due to the increase in intrathoracic pressure. This haemodynamic effect with reduced blood flow to the brain can then be the decisive factor for death by asphyxiation (see 'Compression of the thorax and pneumothorax').

### Impairment of respiratory movements

Extensive rib fractures are a frequent cause of obstruction of thoracic movements. If the chest wall including the pleura costalis is injured, there is an open pneumothorax. The intrathoracic negative pressure is lost and the lung on the affected side of the chest can collapse to a greater or lesser extent. As a result, hypoventilation of varying intensity occurs, which can even result in death by asphyxiation. A double-sided open pneumothorax is particularly dangerous and can seldom be survived without immediate medical help. Closed pneumothorax can occur as a result of punctures of the lungs by fractured ribs. In this case, the inhaled air penetrates into the affected half of the chest and increases the negative pressure. The consequences are identical to those described for open pneumothorax.

Restrictions of breathing movements can also occur in the course of poisoning, such as in the case of morphine intoxications as a result of the attenuating effect on the respiratory centre.

Very overweight individuals can have severely restricted breathing movements not only in abnormal body positions but also in prone positions.

### Reduction of gas exchange in the alveoli

Intense pulmonary oedema inhibits gas exchange in the alveoli. The same applies to damage to the surfactant factor, which may result from inhalation of toxic gases. Water also affects the surfactant factor so that hypoventilation can occur. This plays a role in the treatment of near-drowning cases.

## Compression of the neck

Cerebral ischaemia or haemodynamic effects with reduced $O_2$ supply to the brain are caused by the following factors:

- Compression of the cervical veins.
- Compression of the cervical arteries.
- Triggering of the carotid sinus reflex.

In addition, the haemodynamic cerebral deficit in neck compressions can be increased by hypoventilation (see 'Obstructions of breathing: Hypoventilation').

Different forms of violence against the neck are particularly common in the practice of forensic medicine. The pathophysiological consequences differ considerably in some cases.

### Compression of the cervical veins

The blood is drained from the head and neck region via the venae jugulares and the vertebral venous system.

The venae jugulares lie relatively superficially in the anterior soft tissues of the neck, especially the venae jugulares internae, which have the largest diameter. The internal jugular vein can be narrowed or even closed during neck compression. According to Yamasaki et al. [65], the cross-section of the thin-walled veins can already be completely compressed with a weight of 1–2 kg. With such compressions, the deeper-lying carotid arteries, which have a thicker wall and a considerably higher blood pressure than the veins, can still be completely continuous or only slightly constricted. This pathophysiological constellation occurs most frequently when the neck is subjected to force.

The venae vertebrales consist of an inner and an outer plexus. The inner system runs in the spinal canal and cannot be compressed by strangulation. The outer part of the venous plexus is located in the vertebral muscles and apparently has a larger cross-section than the venae jugulares. However, compression of the neck can significantly impede blood flow from the brain. If the impairment of the blood flow is greater than the reduction of the inflow, a considerable passive hyperaemia develops above the compression. In this case, considerably more $O_2$ is withdrawn from the accumulated blood than normally, so that cyanosis develops in the head and neck region. If the obstruction persists, oxygenated blood cannot enter and cerebral hypoxia occurs. Due to the suppressed cleansing function of the blood, mainly acidic metabolites accumulate, resulting in particularly pronounced cell damage. In addition, tissue fluid can be expressed, which can lead to swelling of the face.

The capillary pressure is normally about 20–25 mmHg. Passive hyperaemia results in an increase in this pressure, but also in venous blood pressure. The increase in venous pressure is initially intensified by the $CO_2$-induced increase in arterial blood pressure. As a result, rhexis haemorrhages of the capillaries occur. These are punctiform bleedings known as petechiae. Using confocal laser scanning microscopy, Lasczkowski et al. [35] have confirmed on conjunctival petechiae that these are the consequences of capillary ruptures. According to Jarosch [24], venous pressures of at least 30–35 mmHg are required for the development of petechiae. Petechiae occur most frequently in areas where the tissue pressure is low (e.g. in the eyelids) [9].

According to data from Pedersen et al. [46], it can be assumed that at least 10 seconds are required for the development of petechiae. This time is necessary to fill the venous spaces of the head and neck. According to experiments by Jarosch [24], the occurrence of these bleedings would take at least 3–5 minutes. In the event of a sudden increase in venous pressure in the head and neck region (e.g. in the case of severe coughing attacks [58]), petechiae were found immediately, at least in the conjunctiva. There may be temporal differences in the occurrence of bleeding between abrupt and comparatively slow pressure increases in the venous system. In forensically relevant cervical vein compressions, the maximum increase in pressure should generally be regarded as rather slow. On the one hand, the victim's attempts to defend himself or herself often play a role. On the other hand, isolated venous congestion cannot generally be assumed. Simultaneous drop or increase in arterial blood pressure is also important. Furthermore, the simultaneous compression of the larynx or trachea can lead to changes in thoracic internal pressure, which also has an influence on the formation of petechiae. In addition, the fragility of the capillaries and the blood coagulation status of the affected persons are likely to play a role regarding the time until the petechiae develop. Due to these complex influencing factors, which cannot be assessed, it is not possible to conclude the minimum duration of forensically relevant neck compression from the presence of petechiae.

## Compression of the cervical arteries

The oxygen supply of the brain is determined by the supply of arterial blood. Under normal conditions, the blood supply to the brain is about 40–50 ml/min/100 g tissue. About 3 ml $O_2$/min/100 g brain tissue is utilized. This makes the human brain one of the organs with the highest $O_2$ consumption. At rest, it amounts to about 15–20 per cent of the total requirement of the organism. The cerebral cortex has a tenfold higher consumption than the white substance.

At the same time, the brain has by far the greatest sensitivity of all organs to an $O_2$ deficiency. A decrease of the blood circulation rate of the brain by half can usually still be compensated. If the brain blood circulation drops to about one-third of the normal value (i.e. to a value of about 15 ml/min/100 g brain), cell death can occur either within minutes or up to several hours. At values of $\leq$10 ml blood/min/100 g brain-extensive cell destruction develops within 8–10 minutes [49]. If the cerebral blood circulation is completely interrupted, functional and structural brain damage occurs after only a few seconds (Table 6.3).

The hippocampus, basal ganglia, cerebellum and spinal cord are considered particularly vulnerable. Extensive necrotic changes can occur in the cortex [52].

The brain has only extremely low $O_2$ reserves. This is the main cause of rapid damage to brain structures. In normal conditions, the brain's $O_2$ reserves are depleted after about 10 seconds under complete ischaemia.

**Table 6.3 Damage related to the time of complete ischaemia of the brain (according to Kuschinsky [33])**

| Time of complete ischaemia | Damage |
|---|---|
| 4–7 s | No damage<br>Possible visual impairment/'blackout' |
| 15–20 s | Loss of consciousness (fainting)<br>Changes in the EEG<br>Symptom-free survival possible<br>Irreversible cell damage increases with longer ischaemic periods |
| 3–6 min | Irreversible diffuse cell death<br>Post-anoxic encephalopathy, possibly coma |
| 7–10 min | EEG signal fading<br>Breakdown of synaptic transmission<br>Failure of the membrane pumps<br>Brain death |

Cerebral ischaemia also interrupts the supply of glucose. The nerve cells themselves contain hardly any sugar. The reserves of glucose and ATP are used up in the neurons within 5 minutes. The neurons are, therefore, dependent on a continuous glucose supply. Approx. 5.3 mg/min/100 g brain are needed. The energy requirement of the brain tissue is almost exclusively covered by oxidative degradation of glucose for ATP production. An anaerobic metabolism, which is already less than 10 per cent under normal conditions, is only possible for a very short time. In addition, the $O_2$ deficiency leads to disturbances of the membrane potentials, which are essential for the transmission of information in the nervous system [32].

The partial or complete compression of the arteriae carotides communes as well as the arteriae vertebrales is of great importance for the loss of consciousness and eventual brain death. The degree of vascular compression is influenced by numerous factors. It depends not only on the size of the compression forces, but also on the internal vascular pressure. The location and nature of the compressive force also play a role. Data on this problem are almost exclusively available in relation to hanging. For example, the diameters of strand tools have a significant influence on the intensity of compression of the cervical arteries – a finding that has not been taken into account by most investigators. The complexity of the conditions allows only for an approximation of the forces leading to the occlusion of the cervical arteries.

It was already determined in the nineteenth century that the arteria carotis communis can be closed with a weight load of 5 kg and the arteria vertebralis with a load of 30 kg [8]. However, the internal vessel pressures that occurred during these experiments were not specified. These pressure values are also important because they can change considerably during the course of certain neck compressions. Based on animal experiments, it is known that strangulations usually lead to a considerable increase in blood pressure compared to obstructions of the respiratory tract [7,25]. Schwarzacher

[57] found 3.5 kg as the minimum traction force for the arteriae carotides communes and 16.6 kg for the arteriae vertebrales at an internal vessel pressure of 170 mmHg and a typical strand position on the neck in order to achieve a perfusion stop. During complete hanging, Brinkmann et al. [6] found closures of the arteriae carotides communes with tensile forces from 5 kg to 7 kg. Atypical strand positions, for example, with the loop running through the mouth and with knots on the side of the neck, were also examined with an intravascular pressure of 170 mmHg. A load of 5–30 kg of body weight was sufficient in any case to close at least two of the four arteries supplying the brain [51]. Yamasaki et al. [65] experimentally tested atypical strand positions at an intravascular pressure of 130 mmHg. In each case, the strand ran under the lower jaw angle. The closing forces for the carotid arteries were 6 kg, for the vertebral arteries 7 kg. Based on all data, it can be seen that the arteries can be closed when hanging if only part of the body weight is effective on the neck. Experimental ligature strangulation has shown that strand tools with a diameter of 15 mm require twice as much tensile force for arterial occlusion as materials with a diameter of 0.5 mm. Furthermore, the arteriae vertebrales could not be closed even with loads of up to 35 kg when the strand was horizontal. This result could be of importance for the frequently observed blood congestion in the head and neck region of ligature strangulation [6]. From practical experience, it can be deduced that the arterial supply of the head is apparently largely or completely interrupted during complete hanging if the weight of the entire adult body acts on the neck. In these cases, there are often none or only isolated petechiae in the eye region [11].

Available data suggest that not all arteries supplying the brain need to be completely occluded in order to lead to such an intense reduction in blood flow to the brain, which ultimately leads to ischaemic brain death. It should be borne in mind that the compression-induced narrowing or occlusion of the arteries is always accompanied by simultaneous occlusion or at least considerable narrowing of the neck veins. In addition, other pathophysiological factors, such as the carotid sinus reflex, can play a role in lowering the cerebral blood flow rate.

Animal experiments have shown that, after ischaemia, a ‘no-reflow phenomenon’ can occur in brain tissue, so that in certain regions reperfusion does not occur even with normal blood pressure. This is apparently caused by vascular spasms caused by $O_2$ deficiency and perivascular oedema [1]. This pathomechanism of post-ischaemic brain damage is of particular importance when a victim survives a neck compression until unconscious and dies after the termination of the force.

## Triggering of the carotid sinus reflex

The carotid sinus is located to the side of the thyroid cartilage, at the front edge of the sternocleidomastoid muscle. It is formed by a widening at the origin of the arteria carotis interna. Pressure receptors are located in the vascular walls which register blood pressure changes and enable rapid counter-regulation. This prevents major fluctuations in blood pressure. Local compression, which reduces the effective blood pressure on the receptors, leads to tachycardia, vasoconstriction, tachypnoea and catecholamine release (relief reflex). Forensically, however, increases in pressure on the receptors are of particular importance as adverse reactions can be observed because nerval impulses from the wall of the carotid sinus are transmitted via branches of the glossopharyngeal nerve to the medulla oblongata. There, a bradycardia is caused via the vagus nerve within 1–2 seconds [22]. The reflex can be triggered both by local pressure on the carotid sinus and by longitudinal stretching of the carotid artery in this region. The stretching of the vascular wall leads to an immediate drop in blood pressure and to bradypnoea (stretching reflex). The affected persons can pass out as a result of cerebral ischaemia. Such compressions and stretching represent an additional pathophysiological factor due to the reduction of cerebral blood circulation in the case of violent effects on the neck [5]. However, this reflex mechanism is of little importance in the case of hanging, as cerebral ischaemia is primarily caused directly by the compression of the arteries. Deaths of healthy persons that can be attributed solely to reflex events have not been proven [28]. It is extremely rare for the carotid sinus reflex to be hyperactive. Such conditions are called carotid sinus syndrome. The syndrome is seen mostly in people over 60 years of age. As a result, syncopal cardiac arrest is possible. The increased triggering of this reflex was also attributed to arteriosclerotic changes in the carotid artery [16,19,29].

## Hangman’s fracture

The term ‘hangman’s fracture’ was first used by Schneider et al. in 1965 [56] and was commonly used particularly in trauma surgery literature. The term stands for a bilateral symmetrical fracture of the arch of the second cervical vertebral body with dislocation between the second and third cervical vertebral bodies. Typically, the fracture gap runs transversely behind the upper joint surfaces of the axis (i.e. in the pedicle section of the vertebral arch) and continues laterally to both transversial processes including the transverse foramina. The dens axis does not break off. This injury pattern had been described decades before, in 1913, by Wood-Jones in museum spinal preparations of deceased persons after judicial hanging [64]. The victims were apparently executed using the ‘long-drop method’.

Schneider et al. [56] had observed these or very similar fractures in patients after traffic accidents. Williams [63] also found such findings after traffic accidents, as well as after falls. It was found that the injury pattern can be caused by two mechanisms, i.e. hyperextension and distraction of the cervical spine and hyperextension and axial loading.

Hyperextension and distraction of the cervical spine when hanging with 'long-drop' typically causes the classic 'hangman's fracture'. This causes high-grade stretching and bruising of the spinal cord, but also destruction of the medulla oblongata. The latter can also be caused by ruptures of the vertebral artery. This mechanism should lead to death abruptly. It is estimated that the height of the fall corresponds approximately to the body length of the affected person or is somewhat longer to trigger the injury effect [53]. Williams [63] states that judicial hanging was generally practised with a fall height of '6 feet' (approximately 183 cm) and that the position of the knot under the chin may have favoured the development of the injury. Pathophysiologically, this is not a death due to asphyxiation, but an injury to the central nervous system that is incompatible with life. In practice, this finding is not of decisive relevance because the simultaneous ischaemia of the brain also leads to death within a few minutes (see Table 6.3). An exception is a hanging case published by Sköld [59] with a fall height of 2–3 metres, in which a modified 'hangman's fracture' was found. The author does not consider distraction with anteroflexion to have occurred because the highest point of the strand mark was behind an ear.

The 'hangman's fracture' injury is also explained by hyperextension and axial loading decompresses – a mechanism that can be observed, for example, in traffic accidents, falls and jumps from height [36]. In rare cases, hyperflexion with axial compression can also be the cause [56,60,63]. Persons who have suffered this injury mechanism usually have little or no spinal cord damage. The patients almost always survive but show varying degrees of neurological damage or are even free of symptoms after the fractures have healed [60].

Retrospectively, the eponym 'hangman's fracture' is not correct. The term 'traumatic spondylolisthesis of the axis', which can lead directly to death when hanging with a jump or fall from a height, would be better. Nowadays, there are differentiated classifications of different types of 'hangman's fracture' in trauma surgery, which are important for the treatment and prognosis of patients (e.g. according to Effendi et al. [14]).

The upper cervical vertebrae are relatively difficult to dissect and are rarely demonstrated in routine autopsy. In this respect, systematic macroscopic studies on the frequency of 'hangman's fractures' are not available. James and Nasmyth-Jones [23] found six cases (18.8%) of axial fractures in exhumed cervical vertebrae of 32 persons corresponding to 'hangman's fractures'. The vertebrae originated from historical judicial hanging cases. Considerable progress in the determination of the 'hangman's fracture' in deceased persons was only made through use of imaging techniques. Using postmortem multi-slice computed tomography, Hayashi et al. [18] diagnosed one case (3.1%) with an atypical 'hangman's fracture' out of 32 deaths after hanging. Apparently, only in this case had a 'long drop' occurred.

In a 2017 review of clinical medicine [42] it was found that traumatic spondylolisthesis accounts for 'hangman's fracture' in 4–7 per cent of all cervical fractures. Other studies in clinical medicine also contain data on the frequency of cervical spine fractures after hanging or hanging attempts, but without details on the vertebral bodies or their fracture patterns. Martin et al. [39] found 6.4 per cent cervical fractures among 563 patients after hanging or hanging attempts. Salim et al. [53] investigated 63 near-hanging cases and found cervical vertebrae fractures in about 5 per cent of cases. In some studies, no cervical spine fractures were observed [3,26,41].

It should be noted that fractures of the vertebral column were found both in the first cervical vertebra and below the second cervical vertebra up to the fifth thoracic vertebra [48,54]. The findings, however, almost without exception, represent secondary findings with regard to the onset of death.

Based on the published data, it can be assumed that fractures of the cervical spine occur only very rarely during hanging and that this particular form of 'hangman's fracture' is a rarity. No information is available on the development of hangman's fractures without the long-drop method.

### Decapitation

An incomplete or complete separation of the head may occur if even greater distraction forces act on the neck than during 'hangman's fractures' with the long-drop method of hanging. In this case, the hanging death is not caused by asphyxiation.

The decisive biomechanical factor for the separation of the head is the kinetic energy of the falling body at the time of the contraction of the sling. If the tensile load limit of the neck is exceeded, separation results. The effective tensile load depends essentially on the body weight and the height of fall. The greater the body weight, the lower the height of fall required for the injury to occur. As a rule, decapitation can be caused in adults with a fall height of about 2 metres or more. The structure of the strand, especially its diameter and elasticity, has a significant influence on the separation effect. Thin, inelastic wire and plastic ropes promote decapitation considerably. The separation often occurs between the first and second cervical vertebrae. Occasionally a 'hangman's fracture' occurs during decapitation [20,40,44].

## ■ Compression of the thorax and pneumothorax

The decisive haemodynamic effect is conditioned by increase in intrathoracic pressure.

In normal breathing at rest, the intrathoracic pressure in the expiratory position is −4 mmHg. Thus, it is always lower than the atmospheric pressure. On inspiration, it assumes even lower values, especially if there is a relevant increase in respiratory resistance. The negative intrathoracic pressure promotes blood return to the heart, while pressure increases hinder the reflux. The passive hyperaemia that develops at elevated pressure leads to hypoxaemic and hypoxic effects in the head and neck region, as described in cervical vein compression (see ‘Compression of the neck’).

Three mechanisms that can cause pressure increases are the Valsalva manoeuvre, pressure congestion according to Perthes, and pneumothorax.

### Valsalva manoeuvre

It is well known that the intrathoracic pressure is increased by expiratory movements when the glottis is closed. However, such increases in pressure also play a role in forensically relevant constrictions or closures of the respiratory openings or airways (e.g. in aspirations). In these cases, the intrathoracic pressure can reach values of significance $>22$ mmHg [29]. This leads to congestion on the venous side of the circulation. Conjunctival petechiae may occur [58]. The cardiac output is reduced, which additionally reduces cerebral blood flow. The face turns cyanotic. Those affected can become unconscious. X-ray studies have shown reductions in the size of the heart [30].

### Pressure congestion according to Perthes

The high-grade compression of the entire thorax is called ‘traumatic asphyxia’ in English-speaking countries. In German-language literature, the terms ‘thoracic compression’ or ‘pressure congestion’ are used. The symptom complex is also called Perthes syndrome after its first description by Perthes in 1899 [47]. The intrathoracic pressure can assume extreme levels and occasionally be raised by increasing the intra-abdominal pressure. The petechiae then occur not only on the head and neck, but also on the skin of the upper chest region and shoulders. Capillary ruptures occur as a result of the interrupted backflow of blood from the upper body regions to the heart with the arterial inflow still existing at first. At the same time, the largely flapless veins of the upper half of the body, into which the blood is pressed back as a result of the thoracic increase in pressure, are of importance. As a result, the petechiae are particularly intense in many cases and can be confluent. In the conjunctiva a hyposphagma often develops. It is possible that the pathomechanism is intensified when inspiration with reflective glottis occlusion occurs immediately before the trauma. Strong cyanosis develops on the face and neck. If the outcome is fatal, considerable brain oedema can be observed. Hypoventilation due to intensive obstruction of thoracic respiration forms an additional component for the rapidly developing cerebral $O_2$ deficiency (see ‘Obstructions of breathing: Hypoventilation’). Nevertheless, several minutes of compression can be survived [4,17].

### Pneumothorax

Severe forms of pneumothorax are also associated with a largely or complete elimination of the negative intrathoracic pressure. Pressure is equalized between the chest cavity and the surrounding atmospheric pressure. This also disturbs the reflux of blood from the head and neck region. In simple cases of pneumothorax, however, hypoventilation is often the most important symptom of $O_2$ deficiency (see ‘Obstructions of breathing: Hypoventilation’). If, however, a tension pneumothorax occurs, for example as a result of chest wall injuries, the intrathoracic pressure increase can become high enough, as a result of the valve mechanism, that the mediastinum is displaced towards the uninjured thoracic side. The disturbed backflow of the venous blood is then occasionally recognizable by the congested protruding venae jugulares. Petechiae can occur in the head and neck region [37].

## ■ Abnormal body positions

Abnormal body positions usually lead to complex physiological changes [21,38]. The pathomechanisms are influenced by the actual physical posture adopted. Particularly significant are:

- Obstruction of breathing.
- Increase in intrathoracic pressure.
- Haemodynamic dysregulations.

In abnormal head postures with trachea constriction, hypoventilation is the dominant factor, but haemodynamic dysregulation with consecutive circulatory disorders of the brain can also play a role.

In the case of head-down positions, cerebral $O_2$ deficiency is caused by several influences. Breathing in may be difficult due to the hanging arms and tension in the respiratory auxiliary muscles. In addition, the abdominal organs push onto the diaphragm, which further hinders inspiration. At the same time, the intrathoracic pressure is increased, so that the suction effect on the venous blood to the heart is reduced. Following gravity, an unphysiologically large volume of blood accumulates in the head, neck and upper thorax. This blood cannot be transported back to the heart in sufficient quantity. The lack of a muscle pump and the missing venous valves in the upper half of the body facilitate this process. The pressoreceptors for blood pressure regulation can also trigger bradycardia and a drop in blood pressure [10,13].

Even in the forced prone position, death occasionally occurs. Those affected usually defend themselves against the posture and are in a strong state of agitation [61]. Obesity and the influence of medication, drugs and alcohol favour the onset of death. Death in a prone position, in particular, is referred to in English literature as ‘positional asphyxia syndrome’ [45].

When assessing the cause of death in abnormal postures, relevant pre-existing physical ailments and the effects of alcohol and other toxic substances must always be taken into account.

Contrary to other types of asphyxiation, positional death obviously occurs after longer periods of time, i.e. after many minutes or possibly even hours. Experiments on rabbits showed that the animals in head-low position sometimes survived for more than 12 hours [62].

## Adrenaline effects

At rest, adrenaline and noradrenaline are released from the adrenal medulla in an amount of about 70 ng/min/kg body weight. In normal circumstances, the adrenaline serum level is less than 100 ng/L. Exceptional physical and psychological exertion, trauma of all kinds, even asphyxiation, lead to a considerable sympathetic effect. The same applies to fighting and escape situations. As a result, the serum level of both hormones, especially adrenaline, increases many times over. The adrenaline causes a complex adaptation of the body to emergency situations. A rapid increase in metabolism is triggered, whereby a higher performance can be achieved. For example, increases in the frequency and contractility of the heart and improvements in the ability of the muscles to work can be observed. The increase in the organism's performance is accompanied by a considerable increase in the $O_2$ requirement of various body tissues. It should be noted that high concentrations of adrenaline can cause arrhythmias. Apparently, the excessive increase of catecholamines can also lead to changes in the ST range and T wave in the ECG, as well as to an increase in QT time, as has been observed in a case of near-hanging [2]. Cerebral hypoxia can be promoted or even induced as a result of the adrenaline effect. The effects are to be considered to varying degrees for the individual types of asphyxiation. In cases of strong defence by the victims, which can be observed during neck holds or blocking the respiratory passages, positional asphyxia syndrome is of importance. It should be noted that hypoglycaemia, certain drugs and alcohol can additionally increase the adrenaline level [29,50].

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# 7 Crime Scene Investigation

Guy N. Rutty and Frances E. Hollingbury

## Introduction

In their textbook *Suspicious Death Scene Investigation*, Vanezis and Busuttil [16] state:

> 'One of the most crucial, if not the most crucial, aspect of the investigation of a suspicious death is the comprehensive examination of the place of discovery of the body – the scene.'

In the early stages of the investigation of a death, examination of the scene where the death occurred is essential when determining whether the death should be treated as a natural event or something more suspicious. If this initial critical decision is made incorrectly, all that follows will be affected. A scene-trained medical practitioner's input into this early decision-making process can prove pivotal in determining how the subsequent investigation proceeds. However, certainly in England, the number of scenes to which pathologists are being called is declining and the decision as to whether a death is suspicious is often being undertaken by individuals with limited experience and even less training in pathology. At times, homicides may be obvious, but all too often the cause and manner of death are not immediately apparent. Similarly, just because a death appears at first glance to be natural does not mean that that is necessarily the case. Those who have worked in death-scene examination for any length of time will have developed a low threshold for suspicion and will have learnt that, even though it might entail a slightly longer examination, erring on the side of caution from the beginning can prove to be the correct course of action in the long term. Jumping to erroneous conclusions can lead to the loss of evidence and, potentially, a missed homicide.

The purpose of this chapter is to provide an overview of the pathologist's role in crime scene investigation in deaths related to asphyxia. For a wider view on crime scene management and investigation, the reader should consult more specialist, dedicated crime scene reference sources [2,3]. They should also refer to their own local, regional or national protocols and guidelines. It is beyond the scope of this chapter to provide a comprehensive review of crime scene investigation on a country by country basis.

## Scenes

In the context of death investigation, a 'scene' (noun) is defined as a place where an incident occurs or has occurred.

In many mechanisms of 'suspicious' death, such as those associated with blunt trauma assaults or stabbings, it is not unusual for the body to be discovered at a place that is not the actual location of the assault. The result is either a geographically large scene or even multiple different scenes to examine. For example, an individual may be stabbed in a property but then manage to walk or even run away from the address only to collapse some distance from the scene of the stabbing. In our experience, this does not tend to be the case when dealing with deaths due to asphyxia. In these cases, the body is usually discovered at the place where the mechanism resulting in the death was initiated. That is not to say that the death had to have occurred at the site of body discovery: for example, a person may have been ligature strangled at one scene and their body then moved to a separate deposition site. However, in cases of mechanical or chemical asphyxia, consciousness is often lost so rapidly that, without assistance from others, there may be steady progression to cardiorespiratory arrest and then the point of irreversibility without further body movement within the scene [10].

Although asphyxia scenes tend to be more contained, there is still the possibility for the body position to have been altered prior to crime scene investigators or pathologists attending the scene. Members of the public, pre-hospital emergency medical teams and other professionals, such as prison officers, may move a body in an attempt to save the individual's life. For example, when dealing with a case of hanging in a prison cell, although the scene remains confined to the cell itself, the ligature is often cut, and the body moved during resuscitation attempts.

Resuscitation attempts may also change a scene in other ways. For example, during the training of one of the authors in his role as a pre-hospital medical responder he was informed that, if an oropharyngeal airway is blocked with blood, this can be rapidly removed, flicked to clear the obstruction and replaced. In doing this, blood spatter could be produced on a nearby surface which may later confuse the investigating crime scene team. Blood patterns can also be produced by responding personnel walking through blood on the floor of the scene, creating additional footwear marks.

It is becoming increasingly common for bodies to be removed from where they were discovered during resuscitation attempts. This may cause further disturbance of the scene as paths are cleared to remove the individual for medical treatment. Although this does not necessarily negate the value of a pathologist visiting the scene, it will make the task of scene investigation more challenging as the body will not be present for context in terms of its relative position to the environment within which the incident occurred.

Every scene is different and the approach to each scene, no matter how simple or complex, is therefore unique. Natural death and suicides, once realised as such, may require relatively little scene assessment. For scenes of suspicious and homicide deaths there are generic approaches to scene investigation that will be considered and applied. These include methods for scene protection, inner and outer cordons, common paths of approach, single points of entry and exit, floor plating and the wearing of generalized personal protective equipment (so-called scene suites). However, as each scene is unique, it is advisable that a scene-specific forensic strategy is formulated between the Crime Scene Manager (CSM) and Senior Investigating Officer (SIO). It is with these two key scene players that the pathologist will interact.

## The pathologist's role at a scene

We, the authors, are strongly of the opinion that pathologists have an important role to play at a crime scene where a body remains *in situ*. They may also have a role to play at a scene after an autopsy has been undertaken, when establishing what object or surface could have caused an, as yet, unexplained injury.

Before a pathologist attends a scene, it is important that they have undergone crime scene training and understand the roles of the various players at the scene. It is essential that anyone attending a scene understands the principles of transference of evidence and that precautions must be taken to ensure that they do not contaminate or disturb the scene. It is also important to remember that the health and safety of those attending the scene is paramount at all times. Pathologists must not let curiosity get the better of them. Remember: Do not visit the parts of a scene which are not directly relevant to your role in the investigation.

There are a number of roles which the pathologist can undertake at a scene of a crime.

### Establishing the fact of death

If the body has not been pronounced life extinct, the pathologist, as a medical practitioner, may be called upon to do this. In the authors' experience, this does happen from time to time, usually because the emergency services have not entered the scene or because the nature of the body's location or recovery has prevented this from happening.

In certain circumstances – for example, hypothermia, drug and alcohol intoxication, diabetic coma and compression asphyxia – an individual may be found in a low-cardiac output state with shallow, infrequent breathing. It is well recognized that shallow breathing can be missed and that it may be difficult for medical professionals who do not do this regularly to detect a carotid pulse. Cases have been described of individuals being pronounced dead or taken to mortuaries and placed into refrigeration only to subsequently be found to be alive [7,8]. The pathologist should therefore never presume life is extinct and should, if necessary and where possible, follow national guidelines when making this determination. An example of one such guideline is so-called 'ROLE' (i.e. recognition of life extinct) [1].

### Establishing if the death is suspicious

Throughout their training and career, pathologists examine a large number of bodies. This experience can be invaluable for scene examinations, particularly in relation to examining deaths from natural or unnatural, non-suspicious causes. It can be easy to forget that other personnel attending the scene do not necessarily have the same experience and some more 'normal' postmortem findings can be easily misinterpreted. For example, a pathologist may be asked to attend a scene due to initial concerns of the emergency services or police that the death may be suspicious only to quickly reassure them that the blood coming from the mouth is no more than gastric contents or likely to be related to a peptic ulcer, or that the 'bruises' to the limbs are, in fact, the cold-related skin changes of hypothermia. The bizarre behaviour associated with so-called 'hide and die' syndrome can confuse the unwary scene examiner but will be all too familiar to the seasoned forensic pathologist. Conversely, a pathologist may upgrade the level of concern when they identify a subtle mark to the neck or the wrist or ankle areas that suggest the application of a ligature in life (Figure 7.1).

By assisting the police at this early stage of an investigation, pathologists can have a significant influence

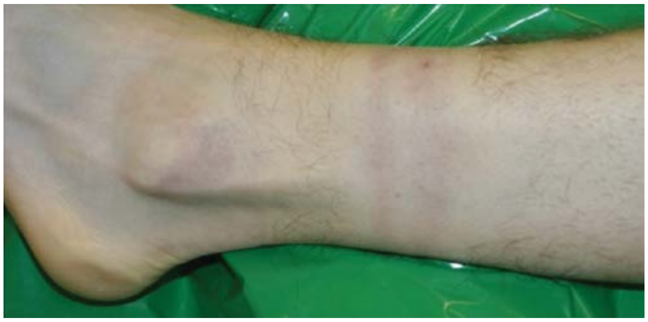

**Figure 7.1** A faint ligature mark around the lower leg of an adult male. The mark was identified on examination by a forensic pathologist at the mortuary and had not been detected by those present at the scene.

on how the investigation proceeds. Establishing that the death is not suspicious will save the police considerable time, resources and money. Similarly, the observation that the death should be treated as suspicious from an early stage will help prevent the potential loss of evidence from the scene and may eventually lead to the conviction of one or more individuals who thought they had carried out the perfect crime.

## Establishing the place of death

It may seem obvious that an individual died where their body was found. However, as with the pronouncement of life extinct, this should never be presumed. For example, a person found hanging from a bridge or tree may have been killed elsewhere and then suspended at the scene to make it look as if they have taken their own life; a body discovered on a beach or in brackish or saltwater may have entered a freshwater waterway and been washed by current or tidal action to a site distant from the place of entry. In the latter situation, a pathologist's knowledge of diagnostic systems such as bacterioplankton polymerase chain reaction (PCR) [9,15] can help ensure that appropriate samples are retained from the scene to assist in determining whether or not this is the case.

Through examination of the body at the scene, the pathologist may identify postmortem changes to the body, or marks or trace evidence on the body that provide an early indication that the scene of discovery is a deposition site and not the site of death. Is the lividity in a distribution that would be expected of the body position at the scene? Are drag marks identifiable on the clothing, skin or heels of the shoes? Loose fibres on the clothing may be left when a body is wrapped in a rug or carpet and then transported to the scene of body discovery. If the person is suspended from a height in a tree, are there any fragments of plant material or injuries to the palms of the hands that would support that the individual climbed up the tree themselves? The more scenes of body discovery someone has attended, the more likely they are to recognize at an early stage that things are not what they appear.

## Establishing the time of death

Pathologists should be aware of the different methods that can be employed at the scene of a death to estimate the time since death. A review of such methods is beyond the scope of this chapter and we suggest that the reader considers a dedicated reference source such as that of Madea [4]. Because every scene is different, an awareness of the different methods available enables a pathologist to advise the SIO when other specialists (e.g. a forensic archaeologist, botanist or entomologist) may be required at the scene.

Initial examination of the body should include identifying the presence of hypostasis and rigor mortis, recording body temperatures and assessing the stage of putrefaction. By using simple generic calculations, such as the Rule of Thumb, a pathologist may be able to provide an early rough estimate of the postmortem interval. This can give the police a starting point and initial time frames in which to search for CCTV or automatic number plate recognition (ANPR) records and target witness accounts regarding the movement of vehicles and people. However, it is extremely important to make it explicitly clear to the police that any scene-based estimation of postmortem interval is no more than that and that investigations outside any estimated time frame must *not* be overlooked or discounted at this stage.

## Evidence recovery

One of the most important parts of scene examination is the development of a forensic strategy for the recovery of trace evidence. As well as a strategy for the scene as a whole, CSMs and the police will compile targeted strategies for specific items or areas of interest, the body being one of them. These strategies are adapted as the investigation progresses, but an initial plan should be in place prior to any examination or movement of the body. Pathologists can play a part in helping develop the strategies and therefore must be aware of the principal types of physical evidence that can exist at a scene [3].

- *Transient evidence.* This is evidence that may disappear or degrade with time, including smells, temperature, imprints and indentations as well as markings such as blood spatter on movable objects (e.g. clothing or furniture) and lividity.
- *Pattern evidence.* This is evidence that is produced when an object or surface comes into contact with another. On a body, this could include blood spatter, clothing patterns, gunshot residue patterns, and the ante- and postmortem injuries.
- *Conditional evidence.* This is evidence resulting from a specific action or event and can assist with reconstruction of events at a scene. From a pathologist's perspective, this can include the location of other items of evidence in relation to the body, such as the location of a potential weapon.
- *Transfer evidence.* Through the principal first described by Edmond Locard, hairs, fibres, dirt, dust, blood, semen, saliva and DNA may all come to be on a body.

In some areas, it is common for the evidence recovery from the body to be undertaken at the scene without a pathologist in attendance, but there are a number of benefits to the pathologist of being present at the scene to assist with this process. While helping with the recovery of swabs, tapings, clothing and personal artefacts, a pathologist can build up an initial impression of the death. This initial external examination and manipulation of the body allows a pathologist to appreciate the appearance of any injuries and the nature of rigor mortis and lividity, unaltered by movement or refrigeration artefact. Even the smell of the body can assist with building up a picture of the death, providing clues as to what the person may have ingested, applied to their body, had applied to their body or been lying in.

The body can be examined, sampled and undressed in the place where it lies. Once trace samples have been collected that do not require manipulation of the body itself, the authors have found it helpful to carefully move the body onto plastic sheeting prior to further manipulation. This helps to capture any evidence that may fall from the body, and it protects the body from contamination from dirt, soil or blood that may be in the immediate vicinity.

During the process of evidence recovery, the entire crime scene team should wear appropriate personal protective clothing not only to protect themselves from the body and the scene but to avoid contaminating the scene with their own fibres, hairs and DNA [11]. Although it is not practical at every scene, it is recommended that as much trace evidence as possible should be gathered prior to removal of the body in order to prevent contamination and potential loss of evidence during the removal. It is becoming less common to use the traditional approach of placing bags over the heads, hands and feet in order to undertake sampling at the mortuary.

### Resource planning

Another reason for a pathologist to attend a scene is to plan for the next stage of their examination. How many deaths are they dealing with from this one incident? Are the bodies fragmented and co-mingled? Are there any health and safety issues such as contamination by chemicals, biological agents or radioactive materials which need to be taken into account? Where is the nearest accredited forensic mortuary? Are there appropriate cross-sectional imaging facilities available? What impact will the arrival of the investigative team have on the routine function of the mortuary? Does the autopsy need to be undertaken immediately or can it be planned for the next working day? All of these and many more questions can be addressed by the pathologist during their attendance at the scene.

### Health and safety

At any scene where there may be a significant health and safety risk, fire and rescue services have primacy for the risk assessment and scene safety. This is separate from scene security, which falls to the police. Pathologists usually have only a limited role to play in terms of health and safety at the scene, although it is important that they pay attention to any risk assessments that have been undertaken. One area where they may be asked to contribute to health and safety assessments at the scene is when the presence of a noxious agent is suspected. If this happens and you have little or no experience in this area, say so. Do not rely on a distant memory of reading something once. There are a number of resources that can be accessed to help in these situations, including national health protection bodies and internet-based resources such as ToxBase. These can generally be easily accessed at the scene on smart phones or tablet computers.

### Body recovery

Pathologists are often asked to help develop the body recovery strategy. The strategy chosen will often be based on previous experience of a similar scene where a particular strategy worked or, perhaps more importantly, where it did not work. Bodies are literally a dead weight which, if they are not handled correctly, can lead to injury to the personnel handling them. It is therefore important that anyone moving a body at a scene has undertaken an appropriate manual handling course. Even with the most careful body recovery process, further injury or disturbance of the body can occur. By staying at the scene for the body recovery phase, pathologists can ensure that any such occurrence is documented, making it easier to interpret any injuries identified at the following autopsy.

## Recording the scene

An accurate and systematic approach to recording the details at a scene is vital. These recordings can be used for briefing the investigative teams, planning scene personnel and forensic strategies, and when considering health and safety at the scene. They are also used in court hearings and even for defending or challenging expert opinions at appeals against conviction.

There are a variety of means of recording a scene and official recording will be undertaken by the crime scene investigation team. However, others, including pathologists, may choose to record the scene for their own reports. It is important to be aware that, whatever the means of recording the scene, any additional record is a disclosable item and must be stored in line with national policies.

### Notes, sketches and plans

By far the simplest way of recording a scene is by using contemporaneous handwritten or hand-drawn notes and sketches. Care should be taken not to contaminate the paper with blood or tissue if one is making such recordings while examining a body. In such circumstances, the pathologist may choose to utilize a scribe to take notes for them. If this is done, care should be taken to ensure that the scribe records what the pathologist sees and says, not what the scribe sees or says. The notes should be signed and dated by the individual making the recording. If not immediately obvious from any hand-drawn sketches, it may be useful to add a note that they are not drawn to scale to avoid any confusion with the official plans drawn up by investigators to capture the scene with architectural accuracy.

## Dictation devices

Manual or voice-activated dictation devices can be used to record notes at a scene. In the past this meant having to store a number of cassette tapes with the case file, but the wide availability of digital devices has reduced this need. Digital dictation enables the files to be stored electronically without taking up precious archiving space. If a digital device is used, the files should be encrypted prior to being provided to an audio typist for transcription.

## Photography

Still photography is still the backbone of all crime scene recording. The images can capture the general environment or detail specific evidential findings or procedures. Any images should be taken at right angles to the item of interest with the inclusion of an appropriate scale. Although pathologists could use their own camera to take still images at a scene, it is easier and often advisable to direct specific images to be taken by the crime scene investigators. This removes the need for multiple sets of images to be exhibited and disclosed.

### Photogrammetry

The process of photogrammetry, a technique for gathering geometric information from still photographs, has been around for as long as modern photography. Today, developments in digital imaging and software have made it possible for photogrammetry to be used to compile a series of digital images into accurate, 3D models of an area or object. This process has been used in areas such as archaeology and engineering for years and is now making its way into crime scene examination and even autopsy and injury documentation. Instead of having to capture a scene with 3D laser scanners, 3D digital models of a scene can be produced using straightforward still photography. The general process is surprisingly simple. Using a camera with a fixed focal length, multiple overlapping photographs are taken of the scene. These are then imported into a photogrammetry software package which puts the photographs together and creates a 3D image [13]. This is movable in the *x*, *y* and *z* planes, allowing the viewer to inspect the scene from any angle as if standing within it. The model can be viewed on a computer screen or even through a virtual reality headset. This latter option should be used with care as, although virtual reality has the potential to allow a jury member to envisage themselves in the middle of a crime scene, it may also have the potential to mislead a jury and thus lead to a miscarriage of justice [16].

## Video recordings

Although pathologists are unlikely to undertake video recording themselves, there are three types of recording commonly encountered in the early stages of an investigation which may assist a pathologist with the interpretation of findings at a scene.

### Body-worn video

Improvements in the quality of easily portable video cameras now mean that police officers, and to a certain extent other emergency services' personnel, often wear body-worn video (BWV). If available, these fixed focal length units, which may also include a microphone for audio recording, can record the moment police officers enter a scene. The footage may reveal the position of a body prior to movement by the emergency medical services, and it can even explain the transference of blood from one room to another if police officers, for example, have inadvertently stood in blood and then carried out a room-to-room search.

### Vehicle video

These same improvements in video-camera technology, and rising vehicle insurance costs, have meant that an increasing number of vehicles are fitted with video and sound-recording equipment. Cyclists are also often seen with helmet-mounted cameras. The footage from these units may capture road traffic incidents as they occur or incidents such as assaults that occur at the roadside. One of the authors, responding to a call in their capacity as an emergency response doctor, has even captured footage of a suspect vehicle leaving the scene of a rural crime. Such footage can be invaluable to the investigative team and can assist the pathologist with the interpretation of injury patterns.

### Scene of crime video

Crime scene investigators may record initial video footage of a scene to produce a basic 'walk-through' for use at early briefings with the investigation team. They may also undertake a more thorough recording of the scene later in the investigation, possibly after the body has been removed. Although not a replacement for actually attending the scene, viewing such footage may assist with understanding the relationship between the body and its environment if for some reason a pathologist is unable to attend.

## Computer-assisted systems

The production of computer-generated graphical representations of scenes of crime and injuries on bodies for court purposes has become a mini industry in itself. The use of photogrammetry to facilitate such work has already been discussed. Laser scanners are another well-established tool for capturing scene information for the production of computer-aided design (CAD) outputs [6]. This work is not usually undertaken by pathologists, but they may be asked to check the work of others to confirm its accuracy.

## Asphyxia-specific scenes

In general, scenes relating to deaths from asphyxia can be categorized into rural (including both land and water), urban outdoors, vehicular and indoors. The reason we choose this categorization rather than considering scenes in terms of the mechanism of asphyxia (positional asphyxia, ligature strangulation, etc.) is that it is often the environment, rather than the cause of death, that provides the investigator with specific challenges in terms of health and safety, personal protective equipment and body recovery. Having said that, the different mechanisms of asphyxia tend to be associated with one or more of these generic environments.

### Rural

Although the countryside may be the scene of discovery of an asphyxia homicide, either as the primary murder site or site of body disposal, it is the authors' experience that it is more common for asphyxia deaths in such an environment to be suicidal in nature, often a result of hanging. (Rural vehicle-related deaths are considered in the vehicle-related scene section below.)

Victims of hanging can be found in a wide variety of rural locations such as woodland, bridges over rivers, and in agricultural buildings. There may be a delay in discovery of the body due to the difficulties of searching for missing individuals in these areas and the individual may have gone to great lengths to ensure that they are not discovered. Always look up when inspecting a scene as it can be easy to miss a body hanging from rafters or high tree branches. Pathologists should be aware of the postmortem artefacts that can be encountered when dealing with these cases. Decapitation may occur when advancing decomposition renders the neck structures no longer able to support the weight of the rest of the body, and there may be evidence of animal predation with scattering of body parts.

The recovery of bodies located in a rural environment can present a number of challenges. If required, the involvement of a suitably trained working-at-height team or water-recovery unit should be considered. This can help to avoid any injuries being sustained by the deceased during recovery (Figure 7.2).

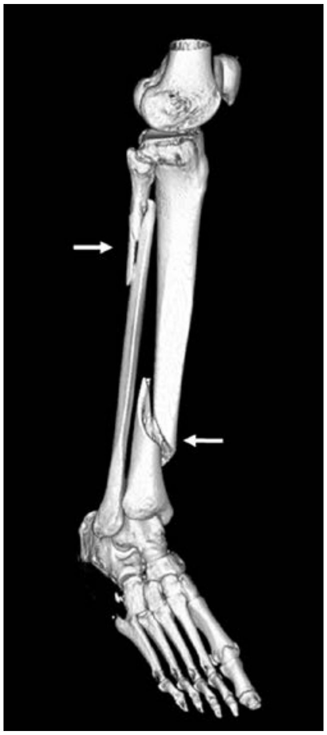

**Figure 7.2** Fractures (arrows) to the tibia and fibula resulting from a body being dropped from a height as it was released from suspension by ligature from a bridge.

### Urban outdoors

Although homicides and suicidal hangings can also be discovered in the urban outdoors setting, other mechanisms of asphyxia, often accidental in nature, may also be encountered. Individuals intoxicated with drugs including alcohol may be found sat against walls or fences, their unprotected airway having become obstructed by flexion of the head in an unconscious state. They may also be found bent across walls or benches, or even trapped while trying to enter or exit a property through a window, resulting in a positional asphyxia. The authors have encountered a case of positional asphyxia in a burglar who became trapped in a chimney flue while attempting to enter a premises (Figure 7.3). This particular case presented a number of challenges when extracting the deceased from the chimney while ensuring that the structural integrity of the building was maintained.

Other mechanisms of asphyxia that can be encountered in an urban outdoors setting, as well as indoors, are the crush or so-called 'compression' asphyxias. These can occur anywhere where crowds gather, for example an outdoor environment such as a stadium or indoors at a nightclub [10]. These incidents may result in multiple fatalities and a disaster victim identification (DVI) approach to the subsequent investigation.

### Vehicles

#### Road-going vehicles

Asphyxia deaths can occur inside or outside a vehicle. When a death occurs inside a vehicle, it is often a result of a vitiated atmosphere/chemical asphyxia or positional asphyxia, whereas those that occur outside a vehicle are often a result of crushing.

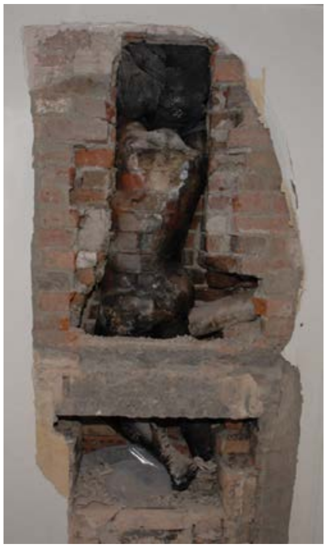

**Figure 7.3** An adult male had become trapped within a chimney flue during an attempted burglary. The position of the arms relative to the head as well as the compression of the chest had resulted in positional asphyxia.

The presence of carbon monoxide from faulty exhaust systems or intentional piping of exhaust into the compartment may result in chemical asphyxia. Suicide by the placement of disposable barbeques or hydrogen sulfide-generating chemicals in the footwells of vehicles has become popular in recent times. In these cases, it is not unusual for those attending such vehicles to be made aware of the potential danger by the presence of notices that have been affixed to the windows of the vehicle by the vehicle occupant [12]. Positional asphyxia can occur following a collision when unconscious vehicle occupants are trapped in an overturned vehicle. More unusually, ligature strangulation, or more strictly decapitation, may be encountered in cases of suicide where the individual ties a ligature around a solid object such as a tree and places the ligature around their neck prior to driving the vehicle away from the object.

Crush asphyxia may occur when the victim becomes trapped under a vehicle and the weight of the vehicle is taken upon their chest and/or abdomen. Although modern vehicle construction reduces the likelihood, crush asphyxia may also occur inside a vehicle. For example, following a road traffic incident, the chest or neck of a vehicle occupant can become compressed against the steering wheel due to a combination of intrusion of the front aspect of the vehicle into the passenger compartment and the driver's seat being pushed forward by the weight of the rear compartment load (Figure 7.4).

### Water-going vehicles

Water-going vehicles can attract a similar variety of asphyxia deaths to road-going vehicles. One more specialized example that can pose a risk to the investigating team is when a death occurs on a container ship. As in grain silos, individuals who have descended to the bottom of a large container may find themselves at risk of asphyxia from a vitiated atmosphere. In such cases it is extremely important to remember that those climbing down into the oxygen-depleted atmosphere to retrieve the individual are also at risk. In addition, phosphine is a commonly used fumigant of cargo holds. It is the responsibility of the ship's master to ensure that the crew is not exposed to the fumigant and there are no stowaways in the area prior to and during use, but it is worth remembering that such chemicals may be encountered if responding to such a scene.

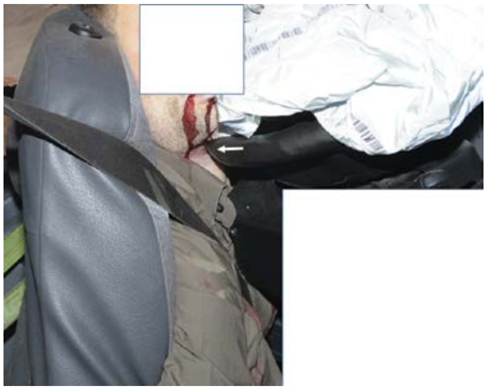

**Figure 7.4** A driver of a vehicle dies at the scene. The steering wheel (arrow) has become horizontally orientated due to vehicle compartment intrusion and the driver's seat has been forced forward as a result of movement of the vehicle's load. This has resulted in pressure being placed on the neck of the driver who is trapped in the vehicle.

## Indoors

The full spectrum of accidental, suicidal and homicidal asphyxia deaths may be encountered in an indoors setting. Even vehicle-related asphyxia deaths, in the form of inhalation of exhaust gases, can occur inside garages. Pathologists will deal with deaths due to plastic bag asphyxia, smothering, ligature and manual strangulation, and hanging. When ligatures have been secured over a door, the body may fall to the ground on opening the door and it may, therefore, not be immediately clear how the hanging occurred. It is always useful to check the tops of doors or beams for evidence of where a ligature may have been. This may also provide evidence of previous hanging attempts. Cases of chemical asphyxia may be encountered in workplaces such as a jeweller's where cyanide is used, and gases such as helium may be used to commit suicide [5].

Another form of asphyxia death encountered in an indoor setting is related to sexual practice and includes so-called autoerotic deaths. Although mainly indoor occurrences, they are not exclusively so. An unusual reported example was a body found in water, originally suspected to be a hanging from a bridge. A subsequent search of the scene found multiple rocks in the water below the bridge which suggested that in fact the individual had died during autoerotic water-based suspension practice which they had undertaken at this site on a number of previous occasions [14].

Autoerotic deaths are usually, although not exclusively, solitary acts. The cases involve more young males than females and often result from compression of the neck following the failure of a release mechanism. The presence of erotic material or professional or handmade implements at the scene may help with determining the circumstances behind the death. Many different everyday items can be used in autoerotic practice and there should therefore be careful consideration of everything at the scene. The authors have encountered the use of a modified vacuum cleaner to aid masturbation and are aware of a case involving a balloon fetish. Electrical stimulation may be part of the process, and this may pose an additional hazard at the scene.

## ■ Summary

Although it is our experience that the number of scenes that pathologists are invited to attend is declining, the pathologist has the potential to make a significant contribution to the examination of a scene. SIOs and CSMs should always consider utilizing the knowledge and skills of pathologists at the scenes they attend. Where a pathologist does not attend the scene, it is important that they are fully briefed regarding the findings at the scene and have access to the scene photographs and/or video footage prior to the autopsy. This ensures that they are able to interpret any pathology identified in the context of the body's position and surroundings at the scene.

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# 8 Normal Anatomy

*Bruno Morgan*

## Background

Medical imaging techniques have been used in forensic investigations for over 100 years. The main modalities are plain film radiography, computed tomography (CT) scanning and magnetic resonance imaging (MRI). Ultrasound imaging is a crucial tool in clinical examination of the neck, but it is less useful forensically so this will not be discussed.

CT uses the differential attenuation of X-ray beams by different materials to create an image. This attenuation is mainly due to tissue density, although atomic number also plays a part. Therefore bones, containing calcium, appear comparatively denser than soft tissues, even if they have similar density, and metals, unless of relatively low atomic number, can cause severe disruption of the image. The appearance of the image can be altered by how the image is created from the original data returned from the X-ray detectors. Basically, this reconstruction balances spatial resolution (edge enhancement) with contrast resolution (lesion detection). High spatial resolution (hard) algorithms are superior where there is already high contrast (bone–soft tissue or air–tissue interfaces) and reduce the impact of metal artefacts, but they considerably worsen detection of differences within the soft tissues themselves. Ideally, the head and neck should be reconstructed using both algorithms (Figure 8.1). Scanning the head and neck twice, at different angles of the CT gantry, can also mitigate metal artefacts from dental implants. This changes the tissues affected by the artefact (Figure 8.2).

The advantage of CT over standard radiographs is the ability to image single 'slices' of the body in *two* dimensions, making a 3D representation of the body. This avoids the problems of overlapping structures, which considerably reduce the diagnostic ability of radiographs, particularly for soft tissue abnormalities. However, CT still cannot compete with the exquisite detail that can be obtained from radiographs to detect fractures in the extremities, such as the hands. CT has been greatly improved recently, with the advent of multi-slice spiral CT scanners allowing fast scanning and greater 3D image reconstruction ability. However, the greatest impact on forensic science has probably come from the decrease in the cost, and the increased availability of CT scanners.

MRI works on the completely different principle of resonance of hydrogen protons, when stimulated by a radiofrequency pulse in a very strong magnetic field. Although other nuclei will spin (helpful in nuclear magnetic spectroscopy), only hydrogen has enough abundance to create anatomical images. Hydrogen protons exist mainly in water in the body, but also in fat, proteins and all soft tissues to some extent. The great advantage of MRI is not only the ability to create anatomical images of this 'proton density', but also to manipulate the magnetic environment, the way the radiofrequency pulses are transmitted and how the resulting emissions are measured (sequences), to create different 'weightings'. These 'weightings' change the appearance of tissues in many ways. Classic 'sequences' involve $T_1$ weighting ($T_1$-W), which is good for anatomy; $T_2$-W, which is sensitive to oedema; and diffusion weighting, which is sensitive to the ability of water to diffuse across boundaries, and fat or water signal suppression. The multiple ways an MRI image can be created is an advantage, but also a disadvantage because it substantially increases the complexity of the investigation. Furthermore, although quite strong radiofrequency waves are used to stimulate the protons, only very weak radio waves are returned to be measured, making imaging times considerably longer than CT scanning to obtain the same amount of anatomical detail. MRI scanners are inherently more expensive, require more staff time and imaging time, and the strong magnetic field can be hazardous if ferromagnetic objects are allowed within the room. All these factors have made CT scanning the preferred option for forensic investigators, although the particular diagnostic questions asked in the investigation of the very young suit MRI (Figure 8.3).

## General principles

Head and neck anatomy has often been considered one of the hardest areas to master. The complexity of the sensory organs, air intake, food intake and connection from the brain to the rest of the body, all in one of the thinnest areas of the body, makes it hard! There are a few basic rules to observe before attempting to use cross-sectional imaging to investigate a head and neck case.

1. *Have a model of a skull.* To really understand head and neck anatomy I recommend having a 3D representation of a skull, either a real skull, a plastic replica, or even a 3D print of a CT image set (Figure 8.4). This helps understand the complex relationships either side of the

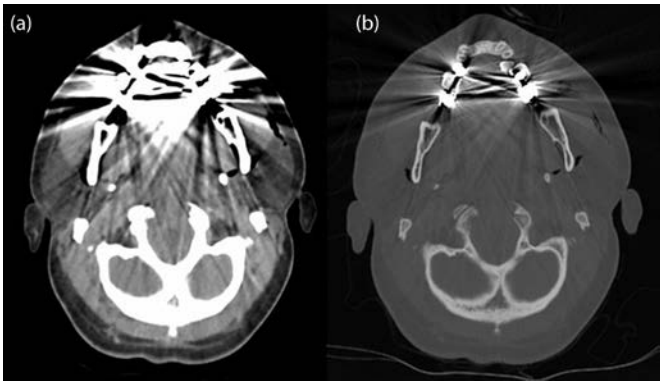

**Figure 8.1** CT scan images reconstructed in the axial plane using a soft tissue algorithm (a) presented using a soft tissue window level and width, and a bone (hard) algorithm and windows (b). The 'soft' algorithm improves 'contrast' to help discern soft tissues, and the bone 'hard' algorithm improves bone 'spatial' detail. There is a severe streak artefact from dental amalgam (heavy metal). Although this artefact is apparently lessened using the bone reconstruction approach (b), and there is improvement in viewing bones, there is no improvement in the ability to interpret the soft tissues.

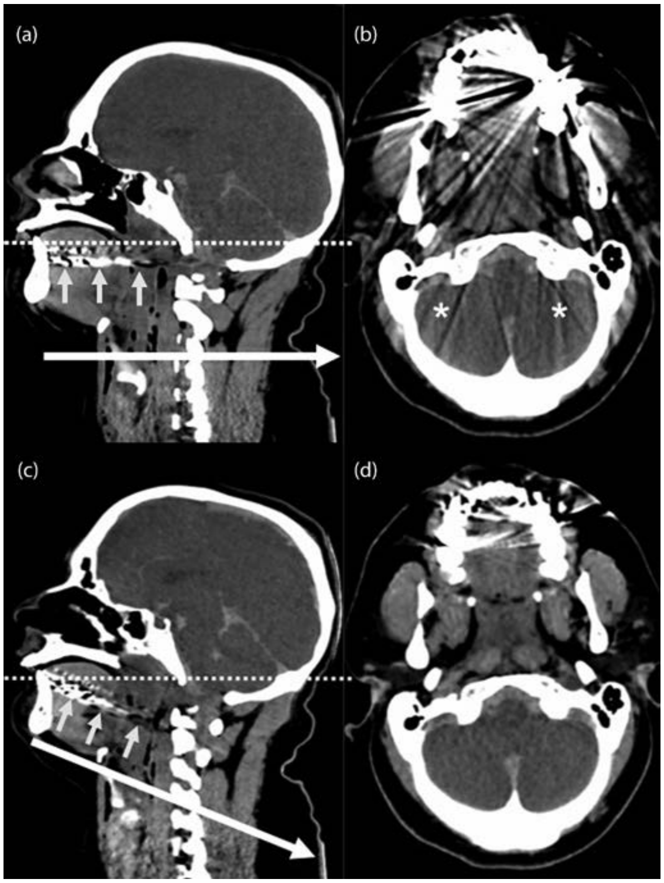

**Figure 8.2** CT image reconstructions in the sagittal and axial plane to show the effect of tilting the gantry. The axial scan plane is shown by the dashed line (a), (b) Images with the gantry in a standard position, so the X-ray beam passes vertically (large arrow). (c),(d) The gantry is angled to shift the X-ray beam angle (large arrow). Images (a) and (c) show that the streak artefact from metal fillings in the teeth (short arrows) follows the line of the X-ray beam. Due to the change in angle, the streak artefact affecting the cerebellum (*) on (b) is not present with the angled gantry (d).

skull base, especially the area around the inferior orbit, inferior orbital fissure and pterygo-palatine fossa.

2. *Do not use imaging alone where direct inspection is possible.* Frequently in clinical imaging, students will diagnose invasion of cancers across boundaries that simply cannot be, and would be discovered to be absurd by simple examination of the oral cavity and throat. This is a tenant of all forensic image interpretation, even with improvements in photogrammetry: Imaging cannot replace thorough external examination of the body.
3. *Reconstruct the images so they are in a straight body plane* (e.g. true axial, coronal or sagittal). In clinical work we go to great efforts to get the head and neck straight during scanning. This is often not practical in forensic imaging. Therefore, always use multi-plane reconstructions (MPRs) with the ability to create oblique planes to get the anatomy straight. This facility creates a new image set in any plane required. If your image workstation cannot do multi-oblique MPR, it is not suitable to interpret images. One of the most useful tools a diagnostic radiologist has is symmetry. If one side is clearly different from the other, then it may be abnormal (Figure 8.5).
4. *Do not always trust symmetry in the neck.* For example, the jugular vein is bigger on the right, cerebral venous drainage not symmetrical and the tonsils can be very different.
5. *Image anatomy atlases are now available in print and online,* but clinical and postmortem anatomy and appearances are different, probably due to the rapid development of oedema (Figure 8.6). There are also changes in tissue laxity, particularly for the oropharynx and airway.
6. *Contrast enhancement is possible and helps demonstrate vascular structures.* However, a good knowledge of vascular anatomy allows large and medium-sized vessels to be tracked without difficulty on non-contrast enhanced scans.
7. *Reconstructions created in 3D are helpful to give an overview of anatomy, quickly identify major pathology and present findings.* However, by necessity, these images have considerably less information available than from scrolling through thin slice 2D CT images. A full review requires inspection of original images, MPR images and 3D reconstructions as an overview.

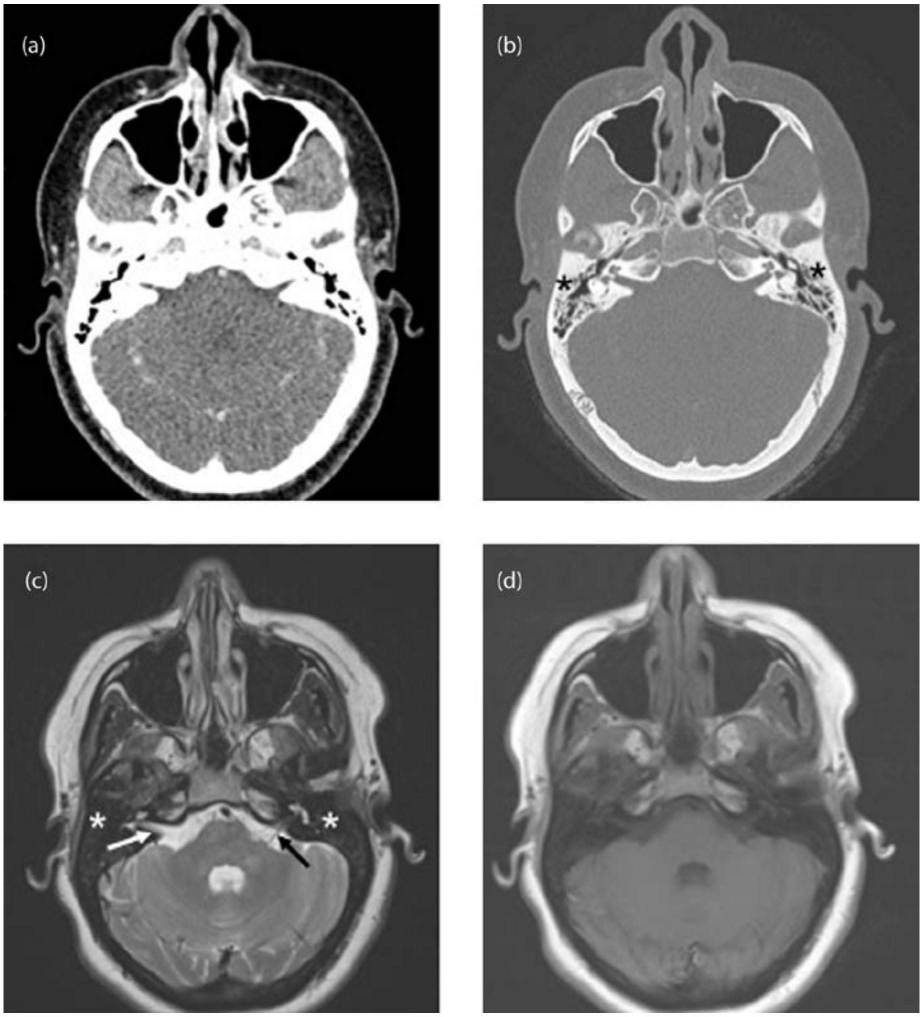

**Figure 8.3** Comparison on CT and MRI at the same level in the same person. Axial CT scan images in soft tissue (a) and bone (b) format, and axial MRI images in $T_2$-weighted (c) and $T_1$-weighted (d) formats. The dark background on the soft tissue CT (a) is a clue that the signal compared with image noise (signal-to-noise) is highest. The CT bone format (b) shows the best bone detail, whereas both MRI images demonstrate better differentiation (contrast) of soft tissues. The $T_2$-weighted image (c) shows fluid as bright, allowing visualization of the 7th and 8th cranial nerves (black arrow) as they pass into the internal auditory meatus of the petrous temporal bone (*).

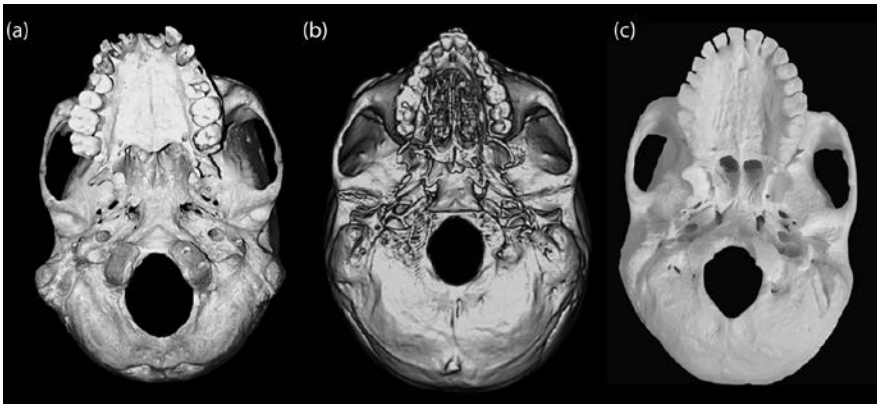

**Figure 8.4** Three skulls. (a) A photograph of a real skull, (b) a snapshot of a 3D reconstruction from a CT skull image set, and (c) a 3D print of a CT image set. It is useful having a skull to hand when studying CT image anatomy, either a real skull or plastic print. This helps to understand the connections through the skull base.

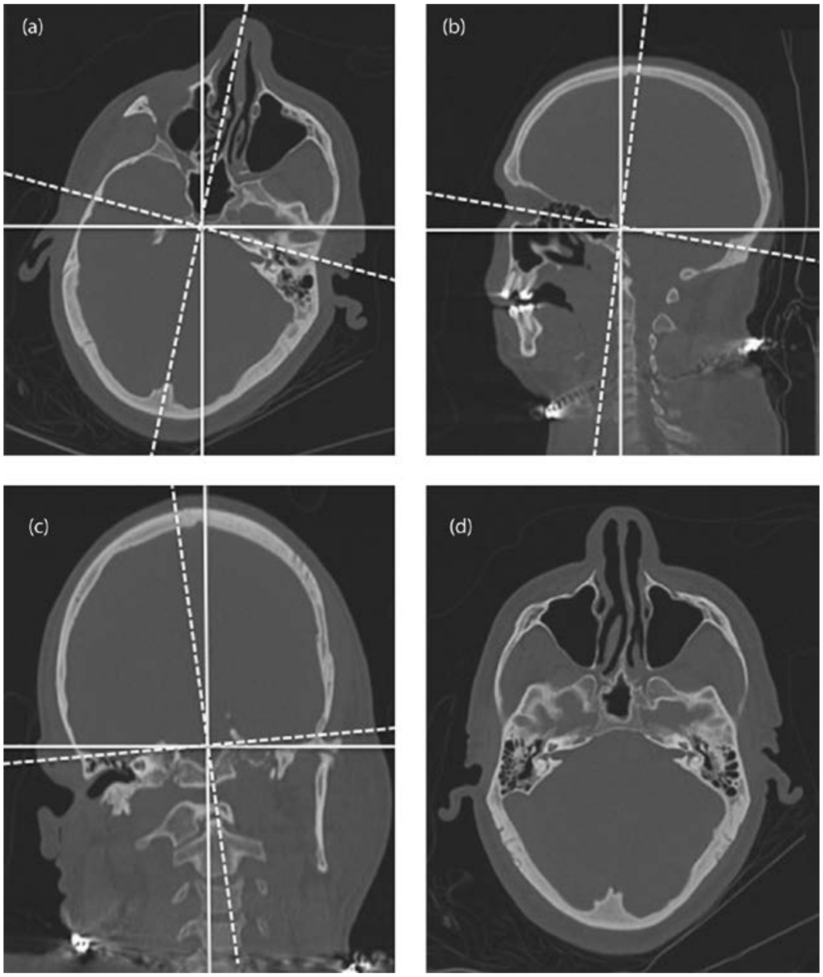

**Figure 8.5** Multi-oblique, multi-plane reconstructions (MPRs). This is a workstation MPR reformat of an axial plane through the petrous temporal bone. Due to the cadaver being in a body bag and rigor mortis, the head is not scanned straight so the original plane (a) is not symmetrical. By altering the reconstruction plane (solid line rotated to dashed line) on the axial (a), coronal (b) and sagittal (c) planes, a symmetrical (true) axial plane (d) is created. This makes pathology, such as fractures, easier to identify.

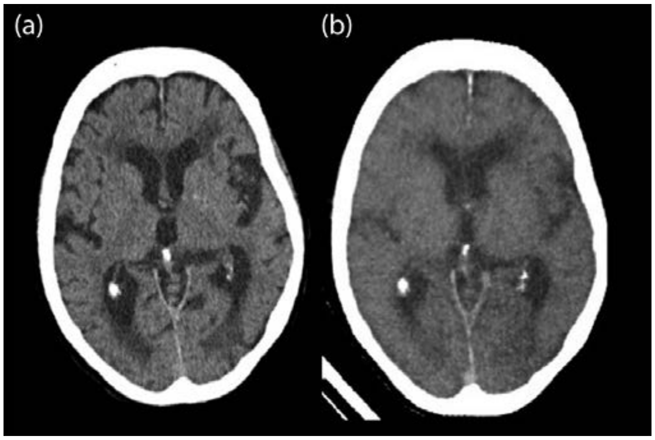

**Figure 8.6** CT images of the brain taken before (a) and 2 days after (b) death. (b) There is blurring of the tissue interfaces and loss of the definition of different aspects of the brain tissue. In a clinical scan this appearance would be consistent with global ischaemia.

## Assessing the head and neck using imaging

Detailed cross-sectional anatomy is beyond the scope of this chapter and is best studied by using one of the many imaging atlases available for clinical practice. However, I will introduce an approach that I find helpful to interpret images.

Start first with bony anatomy. Identifying bony trauma quickly is a big advantage of CT, and identifying trauma significantly affects the interpretation of other ‘soft tissue’ findings. CT scan is generally considered superior at identifying fractures, but MRI is often superior at identifying their consequence. For example, CT can demonstrate fractures of the cervical spine, but MRI can show ligamentous damage, instability and spinal cord trauma. In clinical practice both tests may be used, but this is often impractical for forensic investigation.

## Cervical spine

The atlas with the axis and peg mainly support rotation, and the rest of the cervical spine provides flexion and extension. As stated above, in forensic imaging multiplane reconstructions are required to assess alignment at all levels. 3D reconstructions alone should be used with caution, because if the neck is scanned in a rotated position, they may look dislocated when they just reflect the normal range of movement possible. Ultimately, if in doubt, a rescan with the head repositioned straight may be necessary.

There are many areas to look for fractures, but initial review should cover key principles. First, the spine should be reviewed in the sagittal plane for any steps in the anterior spinal line, posterior spinal line, spinolaminar line and the spinal canal (Figure 8.7). Any step should be treated with suspicion. The soft tissue anterior to the vertebra may be thickened in trauma or infection, but caution should be taken because normal clinical thickness (up to 6 mm at C3) does not always apply in postmortem CT (PMCT), if there is postmortem oedema or the normal cervical spine lordosis is absent.

The spine should then be assessed for facet joint anatomy in both the coronal and axial planes. If the spine is straight, these may be clearly normal on one image, but multiple MPR oblique reconstructions may be required to assess each one (Figure 8.7). Particular attention should be paid to the atlanto-occipital joints (occipital condyles) to check for normal articulation and exclude cranio-cervical dissociation. This is an uncommon injury clinically but common in forensic practice. Normal measurements have

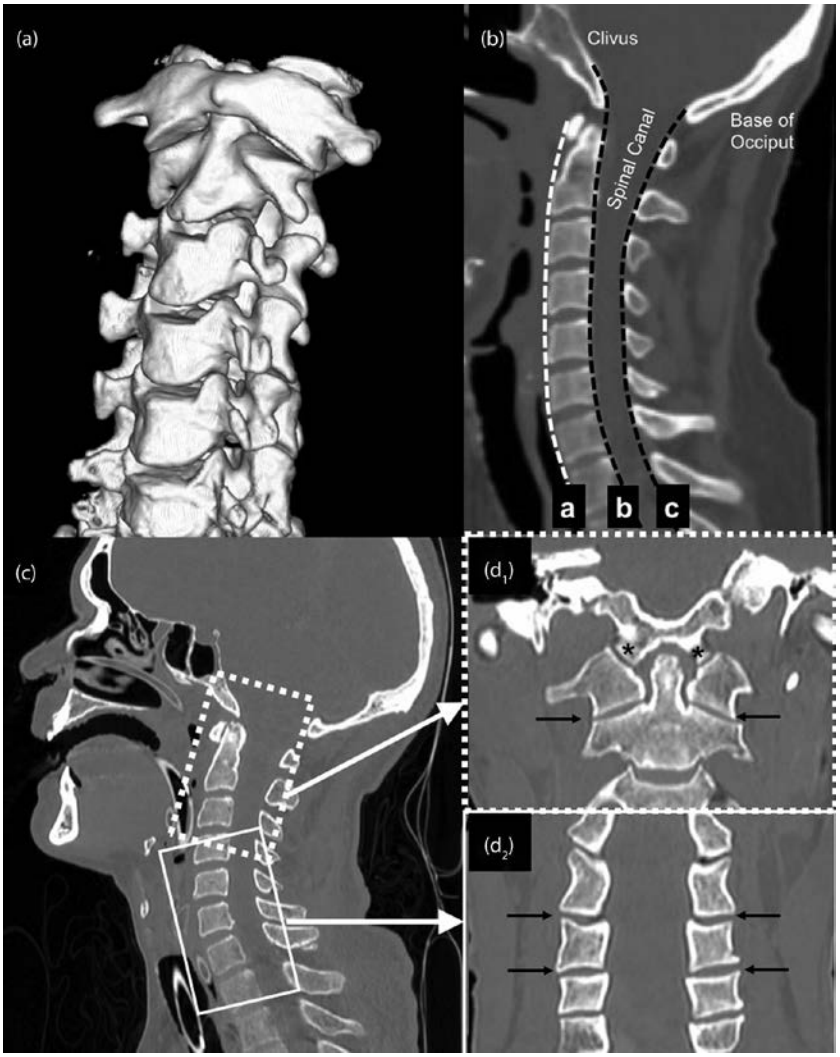

**Figure 8.7** The cervical spine presented as 3D reconstruction (a), and MPR slices (b–d). 3D reconstructions can be used to assess the spine in a manner similar to using a dissected specimen. However, this is time-consuming and will miss subtle fractures. A methodical approach based on MPR is recommended. Image (b) shows the classical vertical stripes that must be in line, the anterior (1) and posterior (2) spinal lines and the spinolaminar line (3). The facet joints (black arrows) can then be assessed axially and coronally ($d_1$ and $d_2$), using different reconstruction angles. Special attention should be given to the atlanto-occipital joints (*), a common fracture in forensic practice, but uncommon clinically (for obvious reasons).

been devised on cervical radiographs for many of these landmarks. However, careful MPR study of the spine and articulations will reveal most fractures and dislocations. Note should also be taken of the vertebral artery passing through foramina in the transverse processes. Dissection of the vertebral arteries is common after neck trauma and can lead to vascular brain injury.

CT is poor at demonstrating the cervical cord and ligaments, which are better appreciated on MRI. In young children significant injury can involve only the ligaments, and MRI would be required to identify it in a spine that has reverted to normal alignment after injury.

## Cranium and facial bones

Proper assessment of the cranium and facial bones is difficult without experience, but the most important thing is to identify normal sutures and other channels such as for arteries and cranial nerves by their pattern of blunt corticated margins and inter digitation. Some sutures can be quite anomalous so identifying their appearance may be more useful than identifying their position. This is more difficult in the developing skeleton, as the reviewer must be aware of growth plates that may simulate fractures. Fractures will have a sharper edge, and can pass through existing sutures (Figure 8.8), although not through recent prior fractures, which can help interpret the order in which fractures have occurred.

## Base of the skull

The base of the skull separates the brain from facial structures, infra-temporal fossa and suprahyoid neck. It is a complex arrangement of four bones, i.e. the ethmoid, occipital (including clivus), paired temporal, and paired frontal bones, all around the complex sphenoid bone. The key is to start with the sphenoid bone, which dominates the skull base of the middle cranial fossa with several aspects to it, including the pterygoid plates inferiorly that link with the facial bones. Laterally, the petrous temporal bone, including the ear apparatus, is well visualized on CT. This links with the squamous temporal bone, which is important because it is thin and fractures easily, which can damage the middle meningeal artery.

Careful scrutiny of the base of the skull is required to detect fractures. Clinically, fluid in the sphenoid sinus and mastoid air cells is a clue to the presence of base of skull fracture, but this is less useful in the elderly and postmortem setting. Lack of fluid in these areas is reassuring.

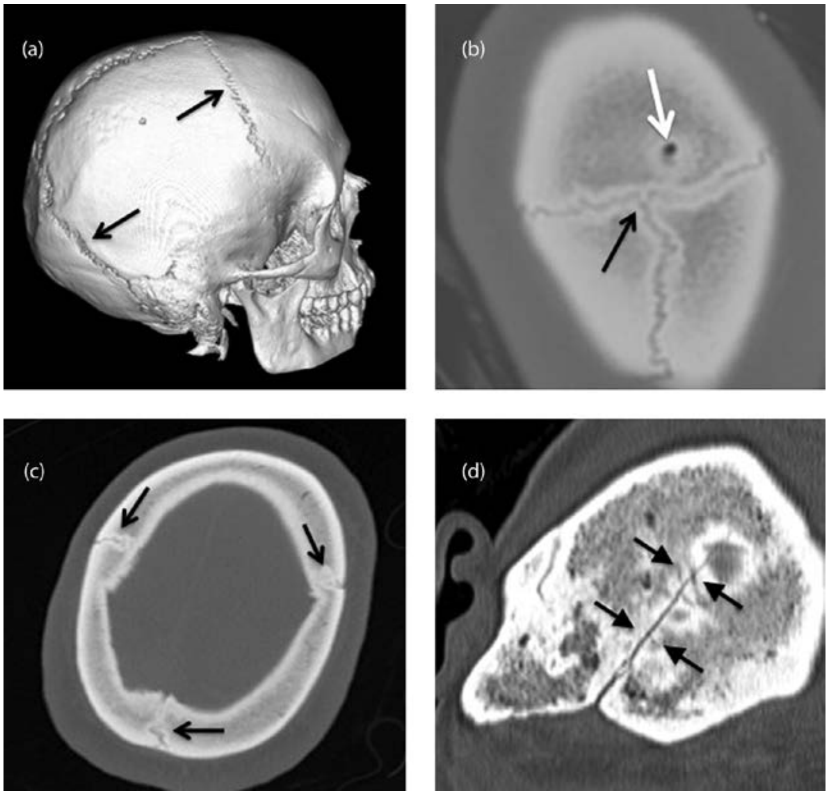

**Figure 8.8** Cranial sutures, foramens and fractures. (a) A 3D 'volume rendered' reconstruction of a skull showing the coronal (anterior arrow) and lambdoid (posterior arrow) sutures. More detail is visible on 2D slices obtained by MPR (b and c) showing the inter digitation of sutures with blunt corticated (dense) margins (black arrows) and a nutrient vessel (white arrow) again with smooth corticated margins. (d) A fracture, which is straight with sharp margins.

The skull base has numerous canals and foramens, which transmit vital structures. Most are visible on thin slice CT scan, but to study their anatomical relationships with the extra cranial spaces requires detailed study, ideally with a scale model of a skull, CT atlas and anatomical textbook.

## Orbit, bones and soft tissue

Due to the high inherent contrast between bone, muscles, nerves and retro orbital fat, CT is often used as the primary clinical imaging tool for anatomical assessment of the orbit. The orbit is contained in a four-sided bone unit with thin walls medially and inferiorly. It is important to look at coronal images to check for fractures of the floor of the orbit, which can involve protrusion of orbital contents into the maxillary sinus below. Interestingly, this weakness actually construes an advantage by causing 'blow-out' fractures rather than damage to the globe itself after direct blunt force injury.

The orbit can be divided into globe, and the posterior space, divided by the extraorbital muscles into the intra- and extraconal spaces. The optic nerve can be clearly seen in the intraconal fat and all structures converge on the orbital apex before passing backwards towards the middle cranial fossa and cavernous sinus.

Clinically, the globe is assessed by direct ophthalmoscopy and occasionally high-resolution ultrasound. CT and MRI are insensitive to retinal and conjunctival haemorrhages, unless they are severe (and visually obvious). PMCT, however, can identify previous surgery, lens implants and dislocated lens. Care should be taken when interpreting trauma to the globe as toxicology may have been taken prior to CT scan (Figure 8.9).

## Facial bones

The paired nasal bones, vomer and nasal concha complex show a lot of variation and asymmetry (Figure 8.9c) so fractures should be interpreted with care after external inspection. The maxilla links via the malar eminence to the zygoma, forming the zygo-maxillary complex (cheek bones) which is important due to the high rate of fractures, and clinically due to the multiple problems, both functional and aesthetic, that these can cause. Generally, fractures are easy to spot, and they are often called tripod fractures as they affect the zygoma, lateral orbital wall and inferior orbital rim with the anterior and posterior maxillary sinus walls.

The maxillary alveoli and mandible hold the teeth. It is worth being familiar with dental anatomy, and taking care to count teeth is important, as missing teeth can be found swallowed or inhaled lower in the neck or chest. Mandibular fractures are normally easy to detect. Clinical scans are generally performed with the mouth shut, but when the mouth is open, as it may be in the postmortem setting, the temporal condyles will normally move forwards (translate), from the condylar fossa to over the condylar eminence. This should not be mistaken for traumatic dislocation.

## Fascial spaces of the oral cavity and neck

Traditionally, the neck is divided into nasopharynx, oropharynx, hypopharynx, larynx and oral cavity. However, with increased use of medical imaging, it is now more common to consider the neck in terms of fascial planes. There is a superficial fascia, a thin fatty membrane enclosing the platysma muscle, and three layers of deep cervical fascia.

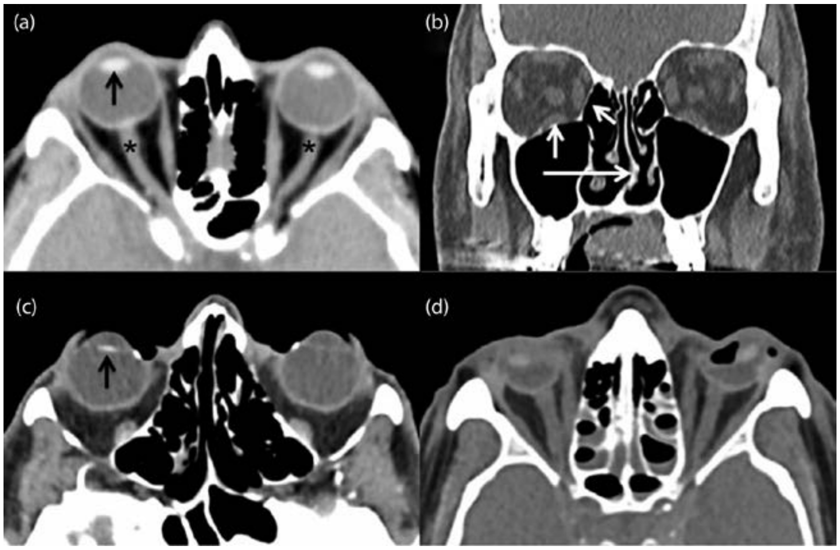

**Figure 8.9** Orbits. Axial (a) and coronal (b) images of the normal orbit. The medial and inferior wall are thin (white arrows), the lens is dense (black arrow) and the optic nerve (*) can be clearly seen in the retro-orbital fat. The coronal images show the optic nerve centrally with surrounding orbital muscles. Absence of the lens or lens implants (c) have a different appearance. Caution should be taken in interpreting trauma in the postmortem setting as the globes may be collapsed, due to previous toxicology testing (d). The large white arrow on image (b) shows a deviated nasal septum.

The fascia themselves may not be easy to see, but they create deep fascial spaces, which are intimately related to the base skull. Dividing the soft tissues into 'spaces' is helpful, and it is an easier way to approach head and neck anatomical interpretation on CT or MRI.

These spaces break down into key spaces. The sublingual (SLS) and submandibular (SMS) space, parotid space, parapharyngeal space (PPS), carotid space, masticator space, pharyngeal mucosal space, visceral space, and the posterior spaces of retropharyngeal space, posterior cervical space and peri-vertebral space.

When assessing the soft tissue spaces, the *parapharyngeal space (PPS)* is a good place to start. It is a largely fatty space making it easy to see on both CT and MRI (Figure 8.10) and extends from the skull base to the hyoid bone. Although it contains little of importance, it is clinically important because of the ways it can be impinged upon by other spaces around it.

The *masticator space* includes the mandible, the muscles of mastication and nerves. Clinically, pathology, such as Infections and tumours, can extend along these fascial planes (elevator effect) (e.g. a tooth infection can extend up the masticator space to the skull base (Figure 8.10)). The foramen ovale of the skull base has been called the 'chimney of the masticator space' as pathology can then extend through it, into the middle cranial fossa.

The paired *sublingual spaces and submandibular spaces* are divided by the mylohyoid muscle and define the floor of the mouth from the mandible to the hyoid bone. They also divide the submandibular and sublingual glands. Anterior dental infections can also extend along the mylohyoid via the SLS space to the SMS space to cause Ludwig's angina and acute airway embarrassment.

## Upper airway

The soft tissues are often divided between the supra hyoid and infra hyoid neck. The hyoid is a forensically important bone at the level of the 3rd cervical vertebra, often little scrutinized in clinical practice. It has a central body with two arms, the lesser and greater horns, although on CT the lesser horn often just appears as small protuberances behind the body (Figures 8.11 and 8.12). It a junction for important swallowing muscles, and the bone can lie asymmetrically if the neck is scanned rotated, due to variable tension of these muscles. Due to fusion and developing ossification, the hyoid bone (and thyroid cartilage) are much more likely to fracture in the elderly,

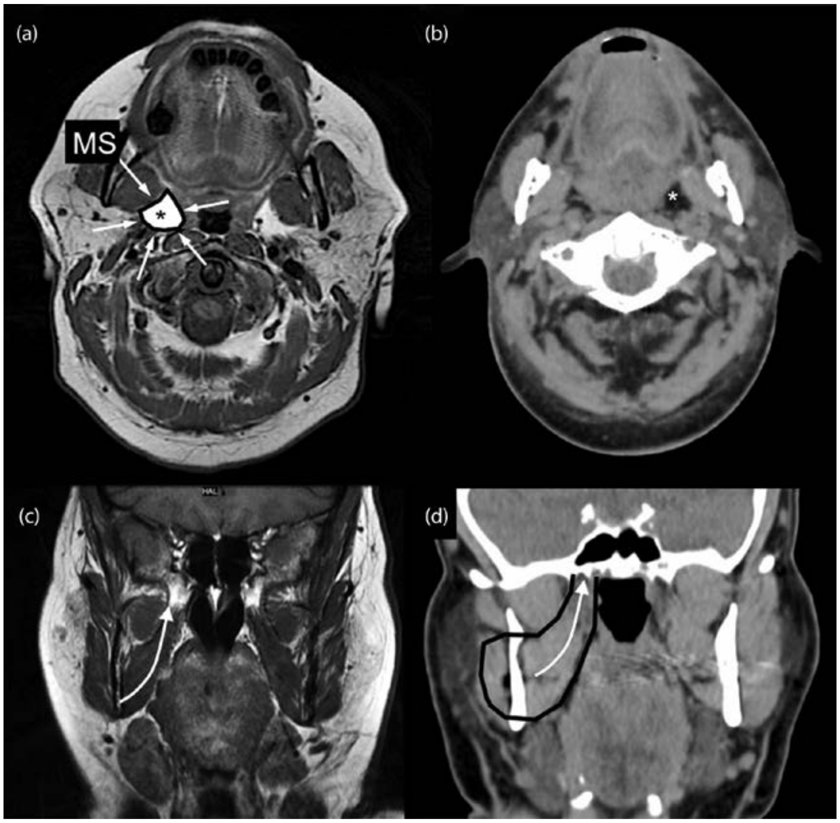

**Figure 8.10** Axial MRI (a) and CT (b) images showing the fat-containing parapharyngeal space (*) (PPS) as white on $T_1$-weighted MRI and black on CT. The PPS is a good landmark as it can be compressed by pathologies in the masticator space (MS) and clockwise the white arrows show the pharyngeal mucosal, pre vertebral (posterior), carotid and parotid spaces. Coronal MRI (c) and CT (d) show the masticator space (black line) and how this can act as an 'elevator' for infection from the mandible to the skull base (white arrow).

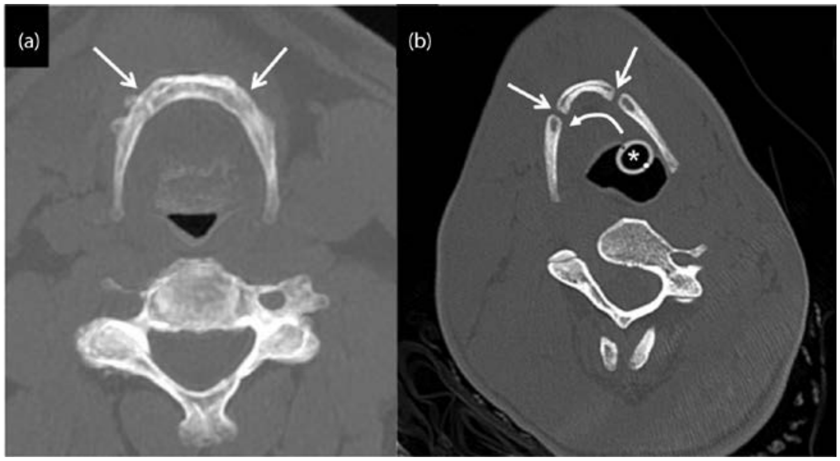

**Figure 8.11** Axial CT images showing an elderly hyoid bone (a) with the body and cornu junction fused (white arrows) and a younger hyoid bone (b) with open joints. Rotation and distortion of the head and neck higher up in (b) are rotating the hyoid bone via muscle attachments (curved arrow). An endotracheal tube (*) is seen in the airway.

due to direct impact, or even traction from the attached muscles.

CT is not as good as autopsy at assessing this bone, but fractures can be detected. This makes an important point between autopsy and CT assessment of bones. Autopsy can spot haemorrhage and oedema around the hyoid to identify the presence of a fracture, while CT relies on angulation, asymmetry and sharp edges. The smaller a bone is, and the greater the variation in normal angulation, the more difficult it is to spot trauma.

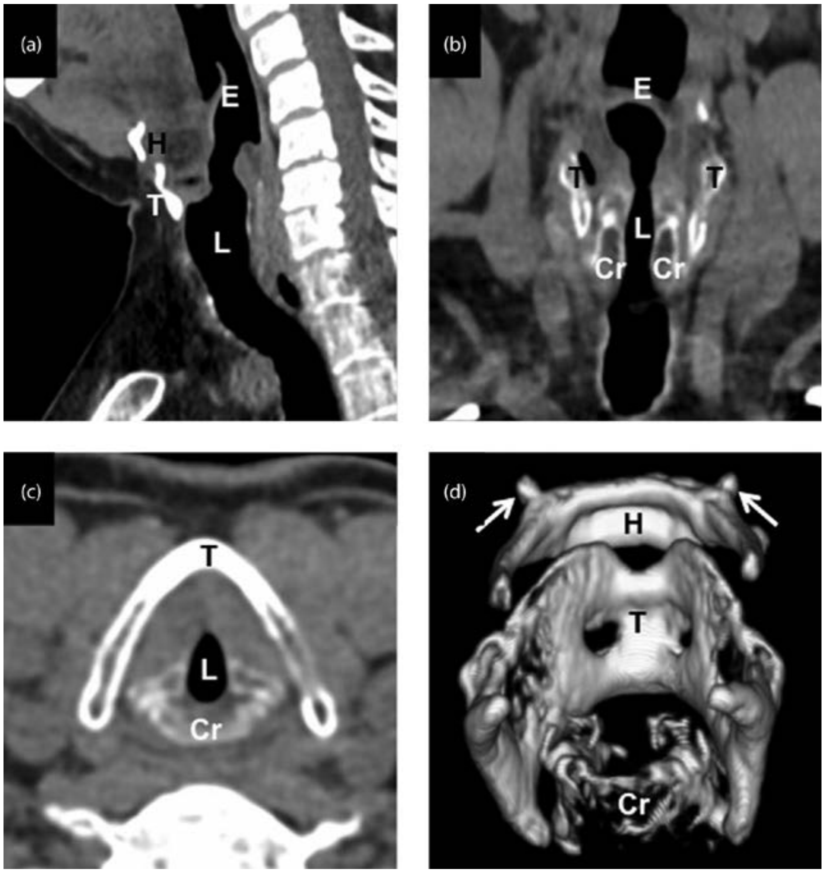

**Figure 8.12** Sagittal (a), coronal (b), axial (c) reconstructions of CT images of the larynx (L). The interrelation of the epiglottis (E), hyoid bone (H), thyroid cartilage (T) and crico-arytenoid complex (Cr) is shown. A 3D reconstruction (d) shows the interrelation from a postero-superior perspective. The lesser cornu of hyoid is seen as small protuberances (arrows).

The upper airway comprises the nasal cavity, nasopharynx, oral cavity, andoropharynx, with the epiglottis protecting as it splits into larynx and hypopharynx. Air patency is apparent on clinical CT (Figure 8.12) but may not be visible on PMCT due to neck flexion on scanning and laxity of soft tissue. Also, the larynx can have opposed or non-opposed vocal cords.

The larynx is usually anatomically divided into the supraglottic, glottic and subglottic regions. The supraglottic region extends from the tip of the epiglottis to the laryngeal ventricle. The glottis is then to just beyond true vocal cords, and the subglottic region extends from the inferior aspect of the true cords to the cricoid cartilage. The variably calcified or ossified thyroid, cricoid and arytenoid cartilages are easily visible. Caution is advised here as this 'irregularity' can make fracture both under- and over-called. This is not a common presentation clinically and would be accompanied by clear symptoms, alerting the reporting radiologists.

CT cannot see small skeletal muscle or mucosal haemorrhages that can be seen on autopsy. This is best done at autopsy. Searching for microhaemorrhages clinically in patients after assault is best done by MRI.

# 9 Pathology

*Silke Grabherr*

Forensic imaging, especially multi-detector computed tomography (MDCT) and magnetic resonance imaging (MRI), have found new applications in forensic pathology [8,11,35]. Today, those methods are mostly seen as adjuvants and used in combination with conventional autopsy. Postmortem computed tomography (PMCT) is often used as a pre-autopsy examination. Thanks to the possibility of looking inside a body very quickly, the subsequent autopsy can be prepared and any specific findings can be viewed even before the body is opened up [30]. This means the autopsy technique can be adapted, and the physician can be attentive to findings that could otherwise be easily overlooked.

In this chapter, findings that are visible with MDCT and MRI are described. The impact of those techniques depends on their sensitivity to detect findings and on the case that is to be examined. MDCT is a technique that is especially useful for cases of traumatic death, as it allows a detailed visualization of the skeletal system with an excellent demonstration of bone findings in two and three dimensions [14,28,33]. However, soft tissue findings, especially small haemorrhages in the muscle tissue, are difficult to identify by MDCT [3,15]. This is why its use for cases of asphyxia, especially in cases of strangulation and hanging, has clear limitations, since, in those cases, the detection of small haemorrhages in the neck muscles are of utmost importance [38].

In such cases, an MRI is much more appropriate, as it allows the investigation of soft tissue, including the detection of haemorrhages [38]. Also, the visualization of brain tissue is better in MRI than in MDCT. This can be of importance to visualize hypoxic lesions in the brain that are common in cases of asphyxia, especially when the patient survived for some time (hours/days).

The various findings that can be viewed with PMCT and postmortem magnetic resonance (PMMR) imaging will be explained according to the type of asphyxia case.

## Hanging

In cases of hanging, MDCT and MRI are indicated, but their sensitivity depends on the tissue in which a pathological finding is located (see Table 9.1).

In cases of hanging, MDCT gives a rapid overview of the inside of the body [38]. First, this excludes findings that are contradictory to the hypothesis of hanging (other causes of traumatic death), and second, it detects findings that indicate hanging. As MDCT is especially useful for investigating the skeletal system and for detecting bone lesions, it can be used to investigate the larynx and pharynx as well as the cervical spine [3,15,30].

Therefore, small fractures of the hyoid bone, the thyroid cartilage and the arytenoid cartilage can be detected and viewed [19,23,38], although it must be borne in mind that very tiny fractures, especially in the cartilaginous parts of the laryngeal skeleton, might only be detected by way of a detailed anatomical section [20]. The possibility of creating 3D images is especially useful if such fractures have to be explained to non-medical persons such as police investigators or prosecutors [28] (Figure 9.1).

3D-volume rendering (VR) software can show the strangulation tool, the surface of the body and any materials on it (Figure 9.2). Evidently, such reconstructions cannot replace the external examination of the body and the strangulation tool. However, such computer-generated images are useful to explain, for example, the position and type of material used or to show the location of the slipknot in a much more impersonal way than by showing a photo of the body. Such images are especially useful if the strangulation material is to be shown and discussed in court.

The most important limitation of PMCT in cases of hanging is the difficulty in viewing lesions in soft tissue such as the subcutaneous tissue and muscles. This is problematic, since haemorrhages in the neck region may be the only visible signs during the autopsy, and should therefore not be missed. Also, in cases of fractures of the larynx, the presence of surrounding haemorrhages should be documented as they are important signs of the trauma vitality. The acute origin of the fracture can, however, be seen more easily with MRI. In fact, fresh and vital (haemorrhagic) fractures show hyperintensity in $T_2$-weighted images (Figure 9.3). $T_2$-weighted MR images highlight fluid accumulations. This makes them an ideal diagnostic tool for a wide range of pathologies [32]. Short Tau inversion recovery (STIR) or $T_2$-weighted water Dixon sequences are suitable for screening purposes because they emphasize the signal from tissues with long $T_2$ relaxation times and fluid accumulations [32].

It is not only direct traumatic lesions of the neck that are detectible by imaging. In all cases of asphyxia, it is important to look for local findings in the neck and for findings related to hypoxia. In cases in which the obstruction of the vessels has led to hypoxic–ischaemic lesions of the brain, the lesions may be visible on an MRI.

**Table 9.1 Findings visible in cases of strangulation and hanging and their visibility in MDCT and MRI**

| Finding | Hanging | Strangulation |
|---|---|---|
| Fractures of the hyoid bone | ++ (CT)<br>+ (MRI) | ++ (CT)<br>+ (MRI) |
| Fractures of the thyroid cartilage | ++ (CT)<br>+ (MRI) | ++ (CT)<br>+ (MRI) |
| Fractures of the arytenoid cartilages | + (CT)<br>+ (MRI) | + (CT)<br>+ (MRI) |
| Haemorrhages in the soft tissue (fatty tissue, paravascular, paralaryngeal space, intramuscular haemorrhages) | ++ (MRI)<br>+ (CT) | +++(MRI)<br>++ (CT) |
| Rope and position of the slipknot | +++ (CT)<br>++ (MRI) | + (CT)<br>+ (MRI) |
| Haemorrhage in the submandibular gland | +(MRI) | ++(MRI) |
| Haemorrhage in the thyroid gland | +(MRI) | ++(MRI) |
| Hypoxic-ischaemic lesions of the brain | + (MRI) | + (MRI) |
| Brain oedema | ++(MRI)<br>+ (CT) | ++(MRI)<br>+ (CT) |

*Notes:* + visible; ++ well visible; +++ very well visible.

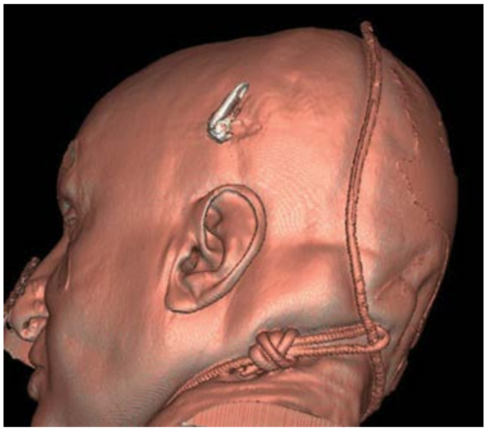

**Figure 9.2** PMCT of a hanging case, VR-3D reconstruction of the body's surface, left view. Visualization of the rope and position of the slipknot.

Until now, imaging methods have been less sensitive than a detailed histopathological examination of the brain following autopsy. However, in some cases with a prolonged survival time after the neck compression event, it is possible to detect morphological findings in the brain due to hypoxia [27]. Such hypoxic lesions can be seen in imaging, especially in MRI and functional MRI, in cases where the patient survived the hanging [2,16,26,40].

The most sensitive imaging examination is diffusion-weighted magnetic resonance imaging (DWI or DW-MRI). This imaging method uses the diffusion of water molecules to generate contrast in MR images. It maps the diffusion process of molecules, mainly water, and distinguishes between whether the accumulation of water within is intra- or inter-cellular. Consequently, it is possible to differentiate respectively between cytotoxic oedema (in which the blood–brain barrier is not disrupted, for example seen in cerebral ischaemia) and vasogenic oedema (in which the blood–brain barrier is disrupted, for example because of an abscess or a tumour).

In cases of large hypoxic lesions, including necrosis and brain oedema, those findings even become visible in MDCT (Figure 9.4). They are regularly seen in cases of those who survived hanging. However, they are usually irreversible and therefore lead to the death of the victim. They then become visible in PMCT, too.

## Strangulation

Just like for hanging, both imaging methods (PMCT and PMMR) can be used for strangulation cases and provide useful information about lesions of the neck and the

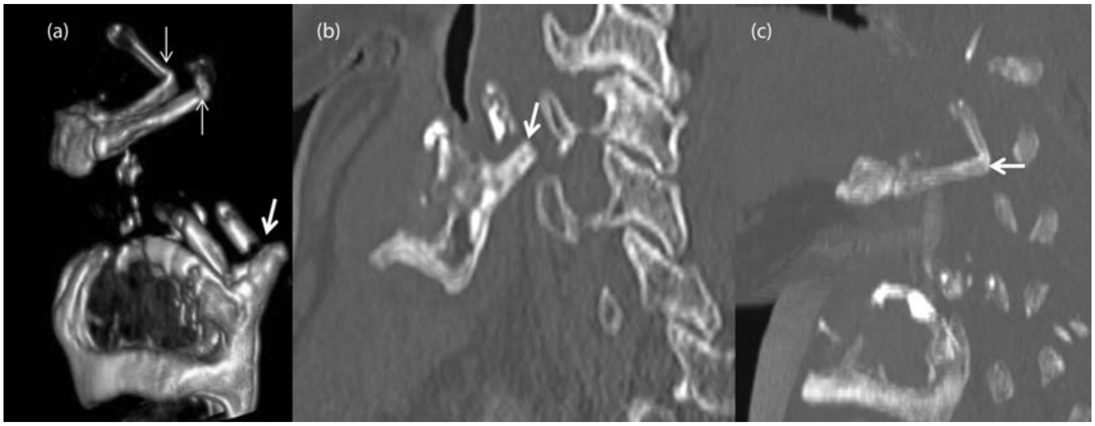

**Figure 9.1** PMCT of a hanging case. (a) Volume rendering (VR) technique, 3D reconstruction, left view. Fracture of the left upper horn of the thyroid cartilage (thick arrow) and of both great hyoid horns (thin arrows). (b) Maximum intensity projection, 3D reconstruction, sagittal view. Fracture of the left upper horn of the thyroid cartilage (arrow). (c) Maximum intensity projection, 3D reconstruction, oblique view. Fracture of the right great hyoid horn (arrow).

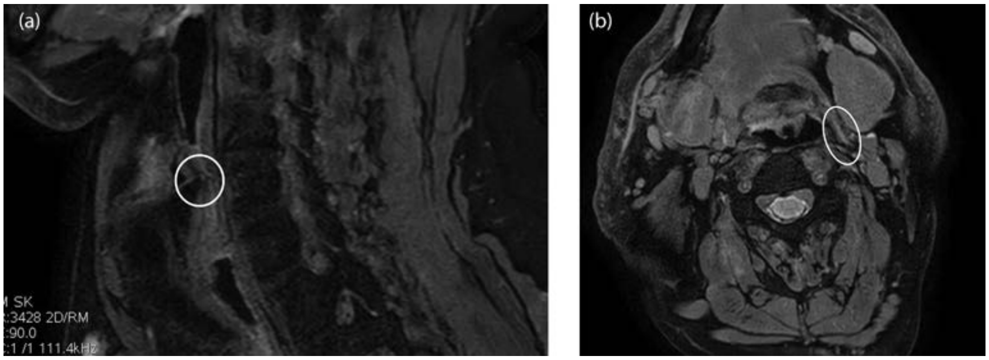

**Figure 9.3** (a) PMMR performed after a hanging case, sagittal view, $T_2$-weighted Dixon water sequence: hyperintensity at the fracture site of the left upper horn of the thyroid cartilage (circle). (b) PMMR performed after a hanging case, axial view, $T_2$-weighted Dixon water sequence: hyperintensity at the fracture site of the left great hyoid horn (circle).

possible strangulation tool. The information gained by these methods is the same as for cases of hanging: a view of the lesions of the larynx using PMCT and the demonstration of haemorrhages in soft tissue using PMMR (see Table 9.1).

There is one indication, however, that must be given special attention. As MRI is an imaging tool that does not use X-rays, it can also be used without harming living patients. This is of special interest in forensics as the examination of victims of survived strangulation is an important issue and part of the daily routine of forensic practitioners. In such cases, it is important to confirm the attack and document any lesions of the neck. It is also of utmost importance to state whether the life of the victim was in danger during the attack. So, a clinical examination has to be carried out, including a detailed anamnesis of the victim's history, clinical symptoms and the actual attack.

Forensic radiological imaging cannot replace this examination as it cannot detect findings on the skin and especially the presence of petechiae. However, it can help to objectify subjective findings such as deglutition pain or respiratory difficulties by showing the morphological substrate such as pharyngeal oedema or compression of the airways by soft tissue haematomas. Additionally, it can detect haemorrhages in the deep soft tissue which are not visible during the external examination of the victim (Figure 9.5). Many papers have investigated the use of MRI to look for any internal lesions in those who survived (mostly manual) strangulation [6,7,29,39]. In fact, lesions such as haemorrhages in soft tissue can be detected.

A very important sign of lesions in the soft tissue of the neck is the asymmetry of structures (Figure 9.6). In addition to intramuscular or intra-fatty haemorrhages,

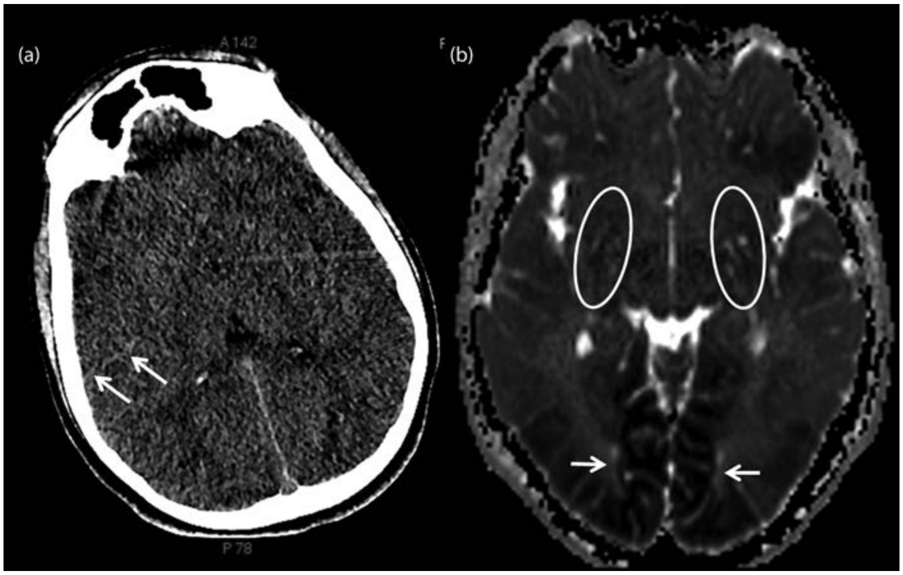

**Figure 9.4** Clinical explorations performed after a hanging case with successful reanimation. (a) Cerebral CT showing a massive cerebral oedema with disappearance of the cerebral sulci and an appearance of pseudo subarachnoid haemorrhage (arrow). (b) Axial view, apparent diffusion coefficient (ADC) sequence: bilateral hypersignal of increased diffusivity in the basal ganglia (circles) and occipital regions (arrows) related to hypoxic–ischaemic changes.

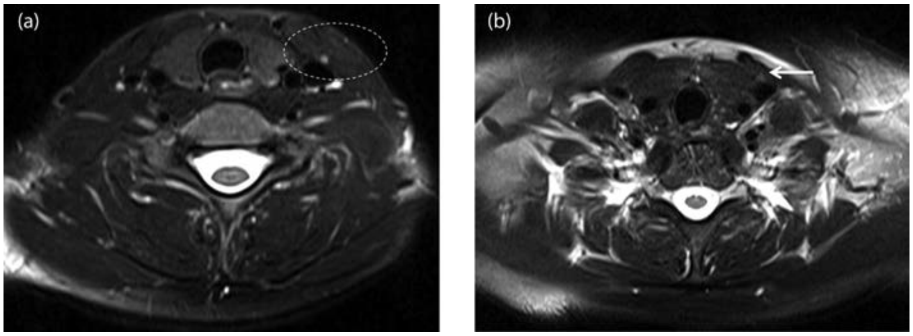

**Figure 9.5** Clinical MRI performed after survived manual strangulations (two different cases). (a) Axial view, $T_2$-weighted sequence with saturation of fat: asymmetry of size of the left sternocleidomastoid muscle associated with an intramuscular hyperintensity (dotted circle) indicating a soft tissue haemorrhage. (b) Axial view, $T_2$-weighted sequence with saturation of fat: hyperintensity within lower part of the left sternocleidomastoid muscle (arrow) indicating a soft tissue haemorrhage.

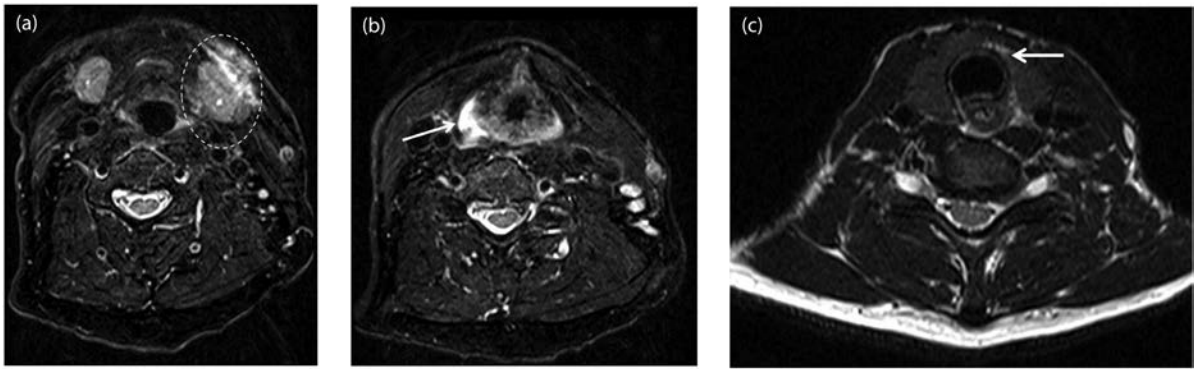

**Figure 9.6** Clinical MRI performed after manual strangulation cases, axial view, $T_2$-weighted Dixon water sequence. (a) Asymmetry of size of the submandibular glands, with a left submandibular gland enlarged, with hyperintensity within the parenchyma of the gland and of surrounding soft tissue (dotted circle). (b) Haemorrhagic infiltration of the right parapharyngeal space (arrow). (c) Axial view, $T_2$-weighted Dixon water sequence: hyperintensity within the left lobe of the thyroid gland (arrow) indicating a soft tissue haemorrhage.

haemorrhages of paravascular or parapharyngeal spaces as well as haemorrhages of the salivary glands and the thyroid gland can be observed as the sequellae of strangulation trauma.

An MRI examination of a living person who survived a strangulation attempt may also be indicated from a clinical point of view [12,13,21,25,34]. In fact, several complications are known and described in clinical literature that can appear after strangulation, even if the symptoms do not appear immediately. Examples of this are delayed post-anoxic encephalopathy [12,34] as well as dissection or thrombosis of the carotid artery [21,25]. There are several imaging methods used to diagnose carotid dissection, including carotid ultrasound, CT, MRI and magnetic resonance angiography (MRA). The imaging study of choice tends to be institution-dependent [37]. Each method tends to have different levels of sensitivity for carotid dissection, ranging from 98 per cent to 100 per cent.

The MR protocol in living persons who survived a strangulation attempt may include sequences with different weighted-contrast images: $T_1$-weighted, $T_2$-weighted sequences, $T_2$ with fat saturation, and STIR, lasting less than 1 hour [7]. The most frequently used planes are axial and coronal. If the Dixon sequence is used, it is possible to deliver up to four contrasts in one measurement: in-phase, opposed-phase, as well as water and fat images. In this case, the protocol is based on the $T_2$ water Dixon sequence and may also be $T_1$-weighted, with and without fat saturation, lasting less than 30 minutes.

## Drowning

The usefulness of postmortem imaging, especially PMCT, for the investigation of drowning has been the subject of multiple studies in recent years [1,4,5,10,17,18,22,31,36]. They defined several findings, visible in PMCT, pointing to a diagnosis of drowning. The results are summarized in Table 9.2.

One of the most often described PMCT findings in cases of drowning is the presence of fluid in the paranasal sinuses

**Table 9.2 PMCT findings described in cases of drowning and their specificity**

| Finding | Specificity |
|---|---|
| Fluid in paranasal sinuses | Low |
| Fluid in trachea and bronchi | Low |
| Frothy fluid in trachea and bronchi | High |
| Fluid in digestive system | Low |
| Pulmonary oedema | Very low |
| Pulmonary emphysema | May be high (not enough data exists) |
| Ground glass opacity | Moderate |
| Bronchospasm | May be high (not enough data exist) |
| Haemodilution | Moderate |
| Dilution of gastric content | Low |
| Sedimentation of gastric content | High |

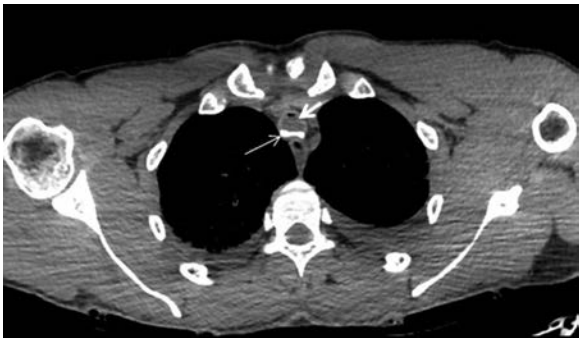

**Figure 9.8** PMCT of a drowning case, transverse image, mediastinum filter. Note the fluid level in the trachea with liquid (thin arrow) and a hyperdense sand-like material (thick arrow) with a horizontal limit between both structures.

[4,5,10,17,18,22,36] (Figure 9.7). In fact, a recently published study investigating the scientific literature to define the state of the art in the diagnosis of drowning using PMCT [36], reveals that fluid is present in the paranasal sinuses in nearly all cases (sensitivity of 70–100 per cent, depending on the study). However, the study also warns that this sign cannot be interpreted as specific, since it is also present in near-drowning cases or even in any other types of case, such as natural death.

Liquid is, however, present in both the paranasal sinuses and in the trachea and bronchi [22]. Again, these diagnostic criteria may be criticised since their specificity is considered low (8% according to one study [22]). To increase specificity, the presence of sedimentation in the fluid can be tested for [31] (Figure 9.8). Also, the presence of a ‘frothy liquid’ in the trachea and bronchi is indicated as a specific sign (100% specificity) according to one study by Levy et al. [22].

Some authors have investigated the presence of pulmonary emphysema by looking for the level of the right hemidiaphragm dome. According to one study [31], its position is significantly lower in cases of drowning than in other cases. These results should, however, be treated with caution, because of the very low number of studies (30 cases only). The same study investigated the presence of bronchospasm. This finding seemed to be related to drowning, according to those results, but, so far, no studies have been performed to validate the results on a larger collective.

Other studies indicate pulmonary oedema in cases of drowning, but none of them describes this finding as a specific finding [4,31,36].

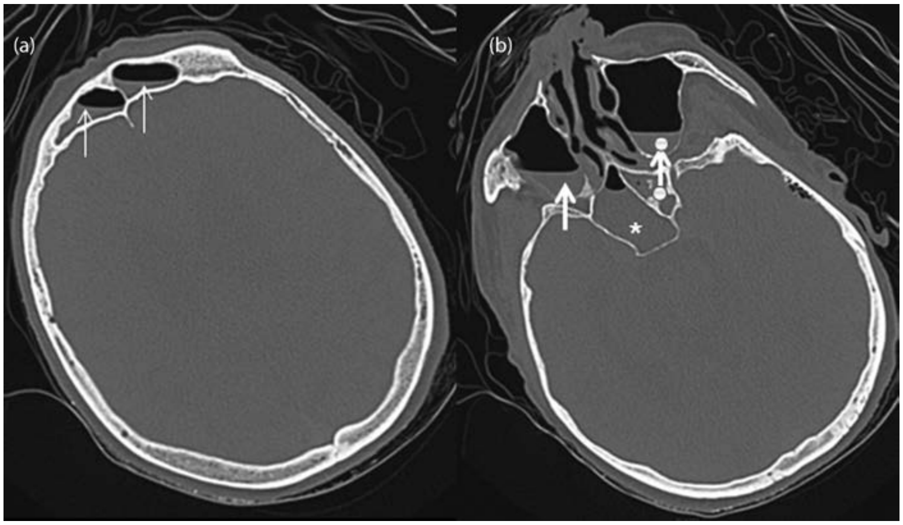

**Figure 9.7** PMCT of a drowning case, transverse images (a, b). Air–fluid levels with the paranasal cavities: frontal (thin arrows), maxillary (thick arrows) and sphenoid sinuses (asterisk).

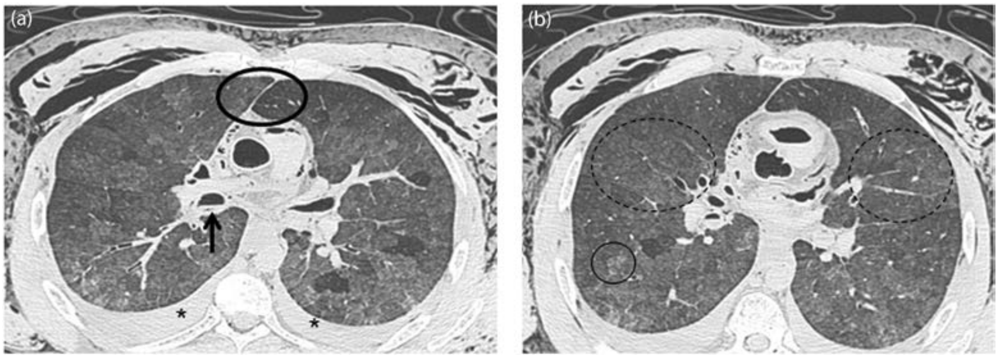

**Figure 9.9** PMCT of a drowning case, transverse images, lung filters. (a) Anterior contact of the lungs (circle); bilateral pleural effusions (asterisks); air-fluid level within the right main bronchi (arrow). (b) 'Tree-in-bud' appearance with a bronchioloalveolar filling (dotted circles) with an overall patchy, 'crazy paving' appearance (both interlobular thickening and a ground-glass appearance) (circle).

One finding regularly described in cases of drowning is the presence of ground-glass opacity [5,10,22,37] (Figure 9.9). These cases usually also show slight pleural effusion and no pulmonary consolidation.

Concerning the gastrointestinal system, a dilution of the gastric content can be seen in cases of drowning that leads to a decrease in mean attenuation (~20 HU according to the paper by Christe et al. [4]. Again, sedimentation of the content is more specific and corresponds to the 'Wydler's sign', visible in a conventional autopsy [5].

As an equivalent to haemodilution visible in conventional autopsy, haemodilution is also described in PMCT. Radiologically, it can be detected by a decrease in the mean attenuation of the blood that is notable in the heart cavities. It is therefore recommended to measure the density of the blood in the left cardiac chambers [1].

## Mechanical asphyxia

There are various scenarios that can lead to mechanical asphyxia. Suffocation due to an obstruction of the airways is regularly seen in cases of accidental death, especially in small children, in the elderly, and in cases of homicide by way of voluntary occlusion of mouth and nose. As already mentioned, imaging is not the method of choice to investigate the most important findings in cases of asphyxia, such as petechiae and small lesions in the soft tissue. This is why it would be impossible to detect haemorrhages in the mucosa of the mouth or small haemorrhages in facial muscles. So, imaging methods have only limited application in such cases, which should be investigated with a detailed external and internal examination, with dissection of the soft tissue, layer by layer.

As already mentioned, however, the effectiveness of PMCT may guide the physician in charge of a case before starting the dissection, by ruling out or detecting traumatic lesions that it is especially important to diagnose in cases of homicides and to find foreign bodies that may lead to the suspicion of mechanical airway obstruction. For these reasons, PMCT has a screening role in such cases. It has several advantages, but medical examiners using this technique need also to be aware of its limits (see Table 9.3).

**Table 9.3 Limitations and advantages of PMCT for detecting findings in cases of mechanical asphyxia**

| Finding | Visibility in PMCT |
|---|---|
| Petechiae | No |
| Small haemorrhages in facial muscles | No |
| Large haemorrhages/oedema in soft tissue | Yes |
| Foreign bodies in airways | Yes |
| Obstruction of airways (e.g. bronchoaspiration) | Yes |
| Pneumothorax | Yes |
| Traumatic lesions thoracic cage | Yes |
| Small haemorrhages in the thoracic cage | No |

By way of a non-invasive investigation, it is possible to find foreign bodies in their original position and to easily estimate the degree of occlusion of the airways, in cases of bronchoaspiration, for example (Figure 9.10).

Bronchoaspiration is important in cases of asphyxia as well as being a vital sign. It is one of the vital signs that are easily detected by PMCT. In fact, the presence of nodular infiltrations in pulmonary parenchyma indicates peripheral aspiration (e.g. blood) in cases of vital trauma (Figure 9.11).

The role of PMCT in cases of traumatic compression of the thorax is even more evident. As mentioned at the beginning of the chapter and in the literature [3,11,14,15,28,30,33,35], PMCT is better than conventional autopsy for detecting traumatic lesions of the skeletal system. Also, it is extremely sensitive in detecting gas and air [9,24], so even the smallest quantities of pneumothorax, air embolism, etc., can be diagnosed. Cases of traumatic mechanical asphyxia are therefore an excellent example of the complementary nature of conventional autopsy and modern cross-sectional imaging. Each technique detects different elements that can be put together for the final diagnosis (signs of asphyxia by external examination and autopsy; signs of trauma by PMCT) (Figure 9.12).

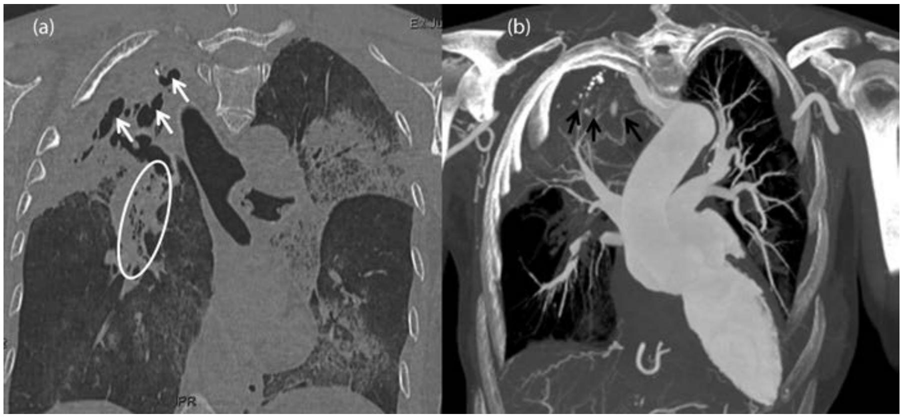

**Figure 9.10** MPMCT of an asphyxia case, coronal oblique images. (a) PMCT: Condensation of the superior lobe of the right lung, with bronchiectasis (white arrows) and obstruction of the bronchial intermediary trunk (circle). At the autopsy, the obstructive material filling the bronchi was clots. (b) MPMCTA, arterial phase: spots of leakage of contrast medium within the superior lobe of the right lung (black arrows).

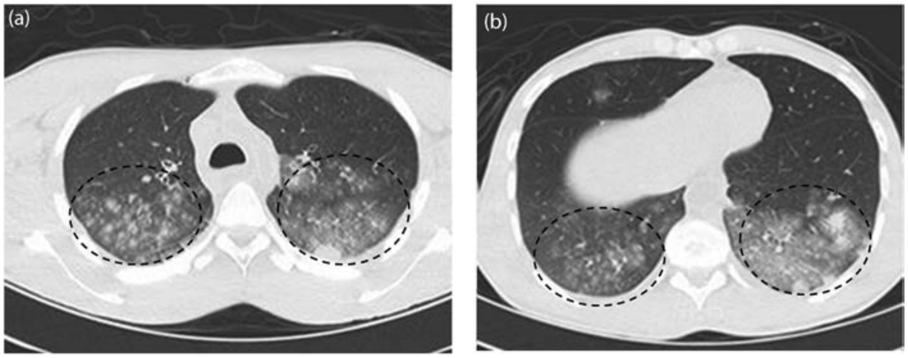

**Figure 9.11** PMCT of a cerebral gunshot case, transverse images, lung filters. Pulmonary parenchymal changes secondary to blood inhalation (dotted circles).

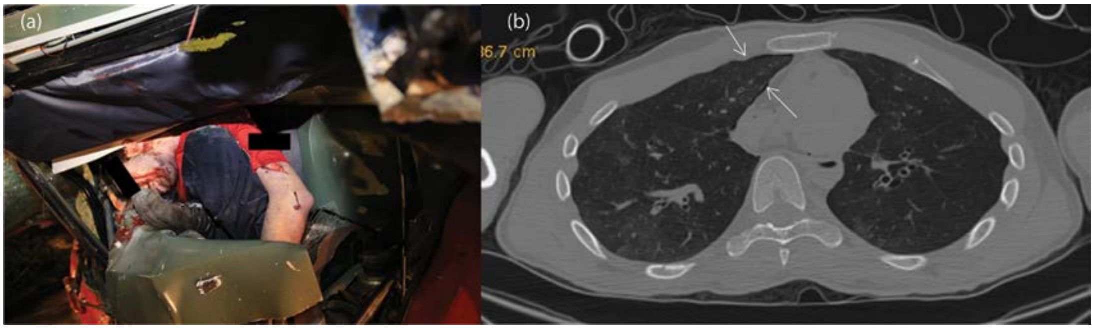
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**Figure 9.12** Case of a train accident. (a) Photograph of the anterior part of the train, with the driver incarcerated in the driving cabin. (b) PMCT (transverse image, lung filter) showing slight right pneumothorax (arrows) and no traumatic parenchymal changes. No rib fracture was noted, although significant compression of the chest was evident.

## ■ Conclusion

Although forensic conventional examination of the body (external and internal) remains the gold standard for examining victims of asphyxia, forensic imaging has a role in the investigations of those cases.

PMCT is an important screening tool for investigating deceased victims, especially to show or rule out traumatic skeletal lesions, including those of the larynx, and to detect foreign bodies in the airways. Its major weakness is the low sensitivity in detecting small lesions in the soft tissue, especially petechiae and haemorrhages in the cervical or facial muscles.

MRI overcomes the limitation of MDCT because of its high sensitivity in showing lesions of the soft tissue. Although its use in forensic medicine is now less widespread than PMCT, it has already been established as a complement to the clinical examination of victims of survived strangulation. It is also sensitive enough to detect hypoxic and ischaemic cerebral lesions, which will increase its importance in investigating cases of asphyxia in the future.

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# 10 Autopsy of Asphyxiation, Suffocation and Neck Pressure Deaths

*Jayantha C. Herath and Michael S. Pollanen*

It is essential to perform a comprehensive autopsy in deaths due to asphyxiation, suffocation and neck pressure. The forensic practitioner and the prosecutor performing the autopsy should be aware of the circumstances of the death prior to dissection and should possess a sound knowledge of the neck anatomy. The pathologist's assistant helping in the dissection should also be well skilled in the dissection technique and knowledgeable in human anatomy. All forensic pathology training programmes and pathology assistant or autopsy technician training programmes train forensic professionals to perform this task well.

One of the major challenges for forensic pathologists is the correct interpretation of observational findings made during external and internal examination of the neck. Often postmortem neck injuries provide considerable difficulties due to anatomical variations of the neck and difficulty in differentiating artefacts from real pathological findings. Sometimes it is difficult to establish the diagnosis of neck injuries as the primary or the contributing cause of death. Often the range of haemorrhage associated with neck compression can overlap with artefacts, giving difficulty in diagnosis in neck injury. That is why it is essential to perform a systemic examination of the neck structures and integrate findings with caution when you provide the final diagnosis.

## Prerequisites and systematic approach for the autopsy

In case of neck injury, a systemic approach for autopsy including the history and circumstances, external examination findings, internal examination findings, ancillary studies and evidence-based opinion is essential.

As prerequisites, it is necessary to have the history and scene information and we recommend carrying out a scene visit before the start of the autopsy. Historical information about the case can be obtained from the investigating police officers or by other death investigating team members such as coroners/medical examiners and medicolegal death investigators. The examination of the body at the scene and the position of the victim at the primary scene are important. A preliminary examination of the victim at the scene can provide important information such as the position of the victim, and postmortem changes including hypostasis, rigor mortis and decomposition. It is also important to find out whether any objects were surrounding the body and/or underneath the neck structures at the scene. It is also useful to note the position of the clothing in relation to the neck at the primary scene. The forensic practitioner can instruct the police officers for necessary photographs to be obtained at the scene. Depending on the time of the body is discovered at the scene and the time interval before the autopsy, the postmortem findings and artefacts are varied. As a result, it is important to take good photographs at the scene even prior to the arrival of the forensic pathologist.

## Postmortem examination

As a systemic approach is necessary for postmortem examination, we recommend that the following steps are followed during the autopsy.

### Alternate light source (ALS) examination

We recommend performing an alternate light source (ALS) examination prior to autopsy. The goal of this procedure is to incorporate best practice using an ALS to identify trace DNA evidence, foreign bodies, latent fingerprints and body fluids in autopsy related to neck injury [2]. There are commercially available ALS examination devices, such as the SPEX CrimeScope®, that can change different wavelengths and perform the light source examination. Police officers, forensic practitioners and forensic science technologists are often trained to perform ALS examination. We recommend enlisting the help of a technician who is capable of operating the device being used in case the operator is not familiar with it. Numerous attempts have been made to recover various trace evidence using ALS examination which can connect the scene, the victim and the assailant. Some skilled forensic scientists and police officers are capable even of obtaining fingerprints from the neck using ALS examination.

### Evidence collection

After obtaining fingerprints from the neck using an ALS examination and collection of any foreign material, we recommend taking swabs from the anterior, lateral and posterior neck surfaces. It is advisable to contact your local crime laboratory and find out the swabbing technique they use. The standard method of collecting evidence using swabs from the neck is done using two swabs slightly moistened with saline. The swabs are placed together and swept in a circular manner from centre to periphery on the anterior, right lateral neck, left lateral and posterior neck skin surfaces. If any visible foreign bodies are present, such as hairs or fibres, they can be collected prior to swabbing and submitted to the crime laboratory for examination.

### Examination of clothing

Examination of clothing is also very important in deaths associated with neck trauma. The relative position of the clothing at the scene and the clothing position at the time of the autopsy should be ascertained. It is also important to make a note about the type of collar of the clothing and the presence of any tight clothing around the neck, such as neck ties, shirt or t-shirt collar or a hooded part of a sweater. With the onset of decomposition, tight clothing can provide artefacts which lead to difficulty in interpretation. It is also necessary to note any jewellery present around the neck as necklaces and other jewellery can also cause postmortem and radiological artefacts.

### Photography

Photographic documentation is an essential part of the autopsy. With modern digital photography, it is not difficult to take and maintain the continuity of photographic documentation from the scene to the final stage of the autopsy. This enables the forensic practitioner to review the images during the various stages of the autopsy. It also provides opportunities to review the process at a later stage, to compare the photographic evidence from the scene and during the autopsy, and also to seek a second opinion about the findings. The photographic evidence makes the autopsy reviewable and can also be produced before courts as evidence. We recommend using the services of a professional photographer with some training in forensic pathology for proper photographic documentation. Some of the subtle findings of the skin can be accurately recorded by using different camera filters and techniques such as infrared photography.

### Radiology

Radiological examination of the neck has become an essential part of modern autopsy. Postmortem radiology now uses digital X-ray, computed tomography (CT) scanning and magnetic resonance imaging (MRI) scanning. It is important to remove any jewellery or clothing before performing the full radiological examination. Most centres are still using plain X-rays or digital X-ray techniques, although the use of CT and MRI scans is becoming increasingly popular in advanced forensic centres. We recommend using CT scanning for any bony fracture and other bony abnormalities of the neck structures and performing MRI scanning for the soft tissue neck structures. It is easy to identify the bony artefacts and 3D reconstruction of the neck structures by CT scanning. CT scan techniques can demonstrate and provide a permanent record of both the radiological artefacts and fractures of the larynx. Fractures of the hyoid bone, superior cornu of thyroid cartilage, thyroid cartilage and cricoid cartilage and their precise location also can be recorded by targeted CT scans.

## Postmortem findings

### External examination findings

There are five major direct and indirect autopsy findings possible during the external examination.

1. Florid petechial haemorrhages of the face (Figure 10.1), conjunctivae (Figures 10.2, 10.3 and 10.4) and mucous membranes (Figure 10.5, inside mouth). Petechial haemorrhages in the eyes can be seen, both palpebral (Figures 10.2 and 10.3) and bulbar (Figure 10.4).
2. Subconjunctival haemorrhages (Figure 10.4).
3. Abrasions on the neck, mostly anteriorly, and at the jawline (Figure 10.6).
4. Discoid contusions on the neck. These contusions resemble the shape of fingertips (Figures 10.6 and 10.7) and are often present on the anterior neck or at the jawline. Sometimes they can be seen on the lateral neck as well as on the back of the neck.
5. Semi-circular/curvilinear/crescent-shaped abrasions. These injuries are seen mostly on the anterior neck and close to the jawline. The directions of the abrasions can vary, but mostly they are directed upwards (towards the face) or downwards (towards the torso) and they are often caused by the fingernails of the assailant. Rarely, they can be caused by the fingernails of the victim during the struggle in an attempt to remove the hands of the assailant.

### Internal examination findings

After careful and stepwise layered dry neck dissection (which will be described later), the following three major findings can be seen.

1. Florid petechial haemorrhages of the muscle sheath and mucosal surfaces of the epiglottis, larynx and pharynx.
2. Haemorrhage of the neck (strap) muscles and the thyroid gland.
3. Fractures of the hyoid bone and laryngeal cartilages associated with acute haemorrhage.

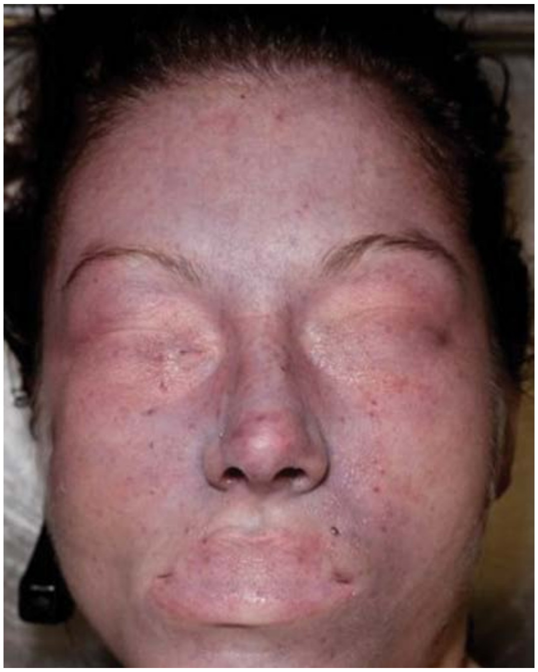

**Figure 10.1** Florid petechial haemorrhages of the face.

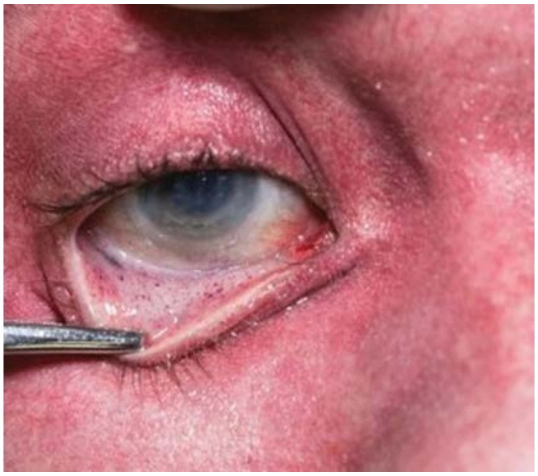

**Figure 10.2** Petechial haemorrhages of the conjunctiva (lower palpebral).

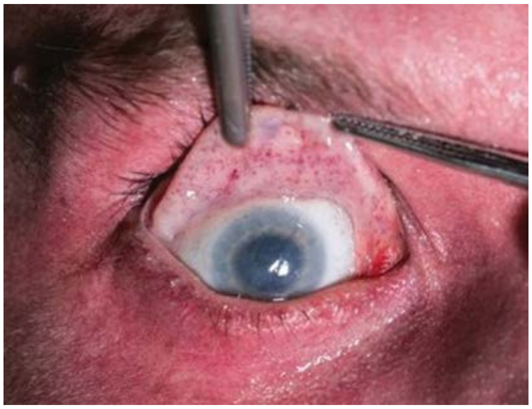

**Figure 10.3** Petechial haemorrhages of the conjunctiva (upper palpebral).

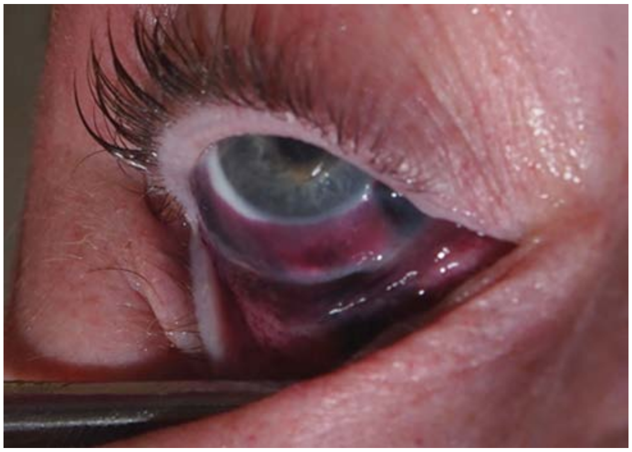

**Figure 10.4** Bulbar and palpebral haemorrhages of the eye.

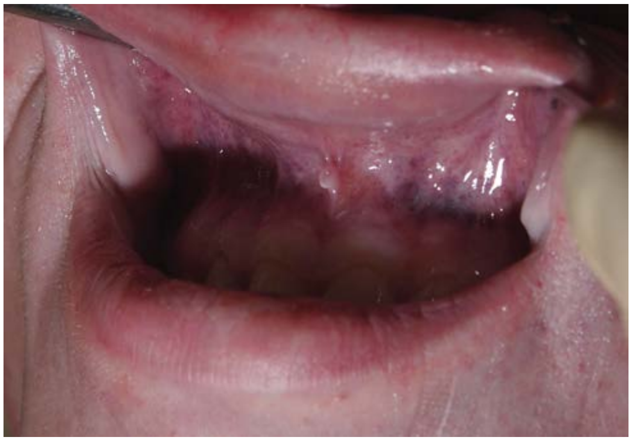

**Figure 10.5** Haemorrhages of the inside of the mouth.

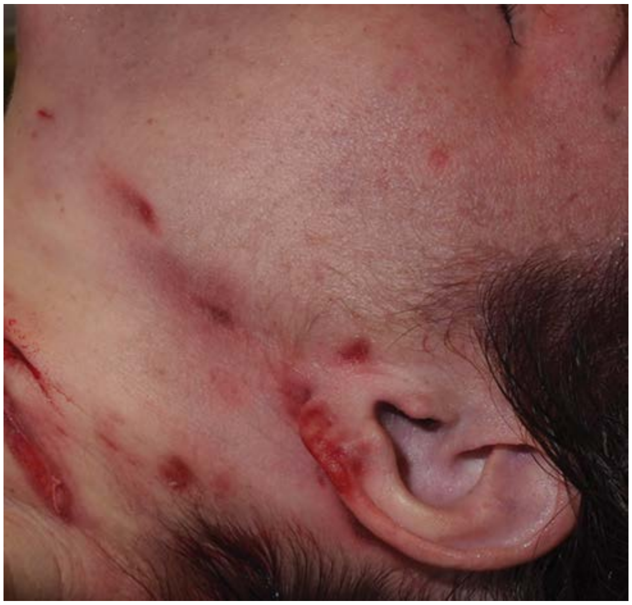

**Figure 10.6** Abrasions on the neck and jawline.

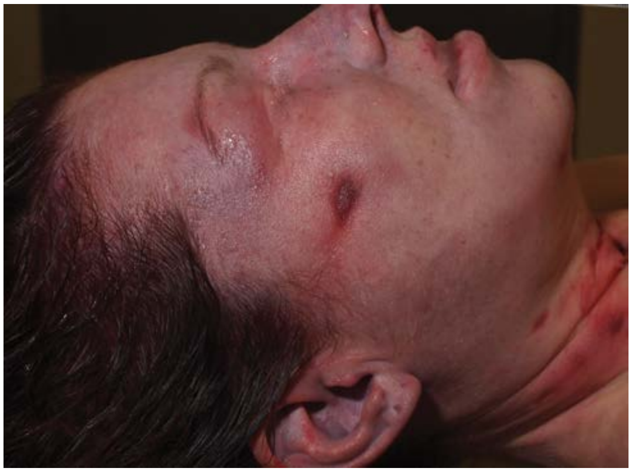

**Figure 10.7** Contusions on the face and neck.

## Ancillary studies

We recommend the collection of trace evidence before the external examination along with a control swab from the buccal mucosa (or a DNA blood spot card from the victim) and blood, urine and vitreous fluid samples to be submitted for forensic laboratory. Samples from the contused strap muscles, soft tissue and fractured bones and cartilages can be submitted for histological examination for ageing of the injury. The DNA material can be extracted from the swabs obtained from the neck and can be compared with a potential assailant. The fingerprint evidence obtained from the neck can also be submitted for comparative analysis. In addition, it is useful to submit nail clippings and a 'sexual evidence kit' in a suspected sexual assault case for comparison of DNA material with a potential assailant.

## Summary and opinion

It is important to understand that the death investigation is a stepwise process. It is essential to follow an evidence-based approach in providing the cause of death and the opinion. A comprehensive death investigation including a thorough review of the history, circumstances and scene examination, and thorough autopsy including layered dissection of the neck and face and incorporating both external and internal examination findings are necessary before concluding an opinion in a case of neck injury. This is often difficult in the presence of a competing alternative cause of death such as a head injury or a stab injury. In the presence of a competing cause of death, the difficult question is to find out the exact contribution of the neck injury to the final cause of death. It is not rare to see a victim who has survived for a short time before receiving a fatal alternative injury. A forensic pathologist is often questioned in court about the possibility of incapacitation by the neck injury before the death occurs.

## Neck anatomy

In this chapter only basic neck anatomy for the purpose of interpretation of neck injuries is described. Please refer to a standard anatomy textbook and atlas for more details. For the purpose of forensic examination, neck structures can be divided into three main parts, i.e. skin with platysma, strap muscles and larynx.

A forensic practitioner needs to know the important landmarks of the anterior neck surface. These landmarks include the chin, lateral angles of the mandible, submental region, floor of the mouth, thyroid cartilage and prominence, hyoid bone, thyroid gland, cricoid cartilage, thyrohyoid membrane, supraclavicular fossae, medial ends of clavicles, mastoid processes, suprasternal notch and clavicular and sternal heads of sternoclavicular muscle.

The strap muscles can be divided into the first (superficial) layer and the second (deep) layer. The platysma is situated between the skin and the superficial layer of muscles. The first layer of the anterior neck muscles includes the sternocleidomastoid, sternohyoid and omohyoid muscles. The second layer of the anterior neck muscles includes the sternothyroid, thyrohyoid and sternohyoid muscles.

The main parts of the laryngeal structures include the hyoid bone, thyrohyoid membrane, super cornu of thyroid cartilage, ala of the thyroid cartilage, median cricothyroid ligament, cricoid cartilage, cricothyroid muscle, thyroid gland, cricoid cartilage and proximal trachea. Once the luminal surface of the larynx is opened, the vocal cords and the mucosal surface can be seen.

## Neck dissection

For anterior neck strap muscle dissection, usual indications are external evidence of neck trauma, strangulation, sexual assault with possible neck trauma or subcutaneous neck haemorrhage upon initial examination.

### Prerequisites

There are a number of prerequisites before the anterior neck dissection is started. It is necessary to remove the thoracic content below the level of the clavicles, remove the brain, and wait for about 10 minutes for the blood to drain from the neck structures.

### Technique

Knowing the anatomy of each layer is critical for performing this procedure. It is advisable to take stepwise photographs and mark the presence of any injury in appropriate diagrams at each step of the procedure. Layered dry neck dissection after removal of the thoracic and abdomen organs and the brain allows any congested blood which can be confused with true haemorrhage to drain from the neck area. The anterior neck structures should be examined layer by layer and any injury should be documented with nature of injury, dimension and distribution and correlation should be made to any injury noticed on the skin surface.

The upper part of the initial Y-incision can be extended up to the tops of the shoulders [1] and reflect the triangular neck flap up over the face (Figures 10.8 and 10.9). The incision from the tops of the shoulders can be further extended up to the mastoid areas making a rhomboid-shaped flap. The anterior skin layer of the neck can be reflected with the platysma. After reflection of the skin and the platysma, the superficial layer of the strap muscles can be observed for injuries and photographed (Figure 10.10). The sternocleidomastoid muscles run along the sides of the neck with the carotid and jugular sheath just underneath. The paired sternohyoid muscles are located centrally. The lower attachments of each of the outer layer muscles can be cut and reflected upwards, leaving the upper attachments intact (Figure 10.11).

The deeper layer is examined *in situ*. The deep layer of muscles is composed of the sternothyroid and thyrohyoid muscles. The sternothyroid muscle is cut at the sternum and reflected upwards, visualizing the underlying thyroid gland and cricothyroid muscles. The thyrohyoid muscle is usually left *in situ*. The neck organs and attached strap muscles are removed *en bloc* with the attached tongue, hyoid bone, larynx and trachea for detailed examination. The larynx and associated structures including tongue, hyoid bone, pharynx, upper oesophagus, anterior thyrohyoid and cricothyroid membranes should be examined in detail (Figure 10.12). The content of the carotid sheath including the carotid arteries, internal jugular vein and vagus nerve should be examined for any injury [5]. The laryngeal block with the tongue, hyoid bone and larynx should be removed using forceps and a larger knife (e.g. PM 40) by making an incision through the suprahyoid, lingual and other muscles attached to the inferior surface of the floor of the mouth, gaining access to the oral cavity. The incision should be made as close as possible to the inferior surface of the mandible. After freeing the tongue, the tip of the tongue can be used to hold the neck structures and a horizontal incision made in the soft palate just above the uvula into oropharynx. Next, the incision is continued into the musculature surrounding the oropharynx, along the prevertebral fascia and the tissue block containing the tongue, hyoid bone, laryngeal structures and pharynx, and the upper oesophagus can be removed after incising the soft tissue inferior to suprasternal notch and inferior surfaces of the medial ends of the clavicles. The laryngeal block can be re-examined radiologically if you are not happy with the images obtained prior to dissection. The neck dissection can be extended to the face to detect and document facial injuries. The margins of the face are extended to midline, leaving the area of the eyes, nose or mouth, depending on the area of interest (Figure 10.13). The spine and the paraspinal muscles and soft tissue can be examined and photographed (Figure 10.14) after removal of the laryngeal structures. In addition to digital photography, conventional pre-prepared diagrams

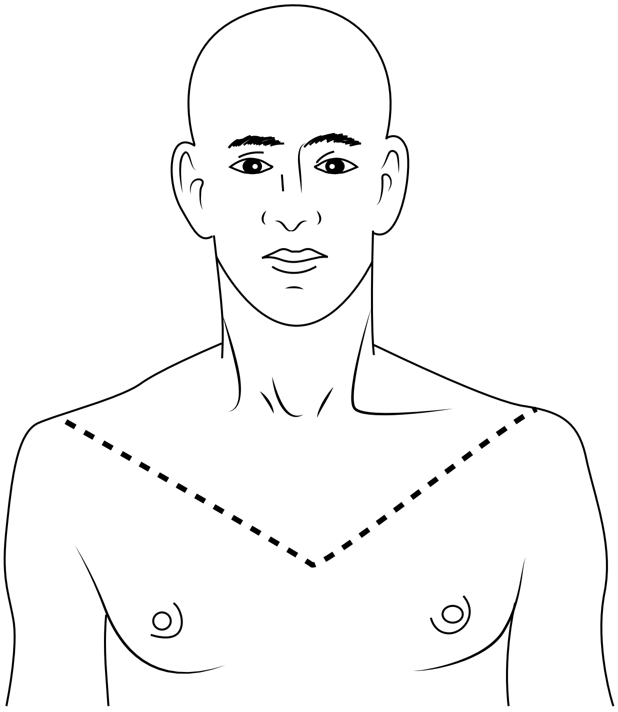
**Figure 10.8** The initial incision can be extended up to the shoulders.

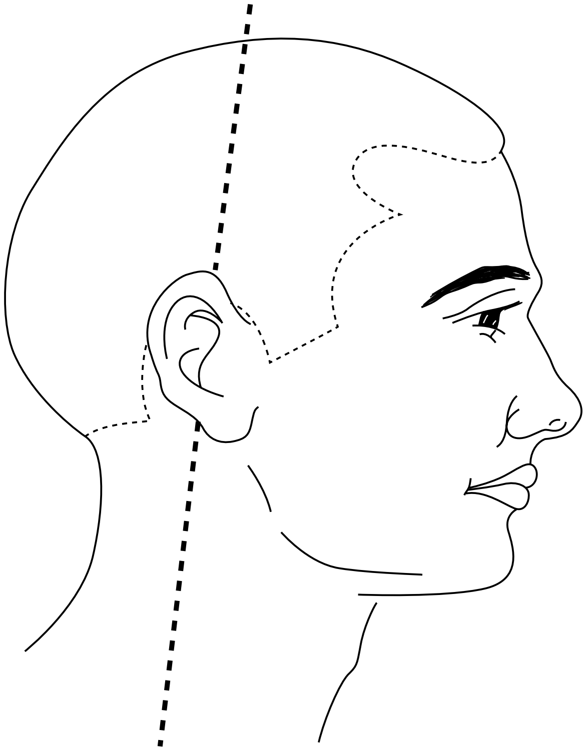
**Figure 10.9** Continuation of the initial incision (shown in Figure 10.8) involving the sides of the neck, passing through bilateral mastoid processes and across the head.

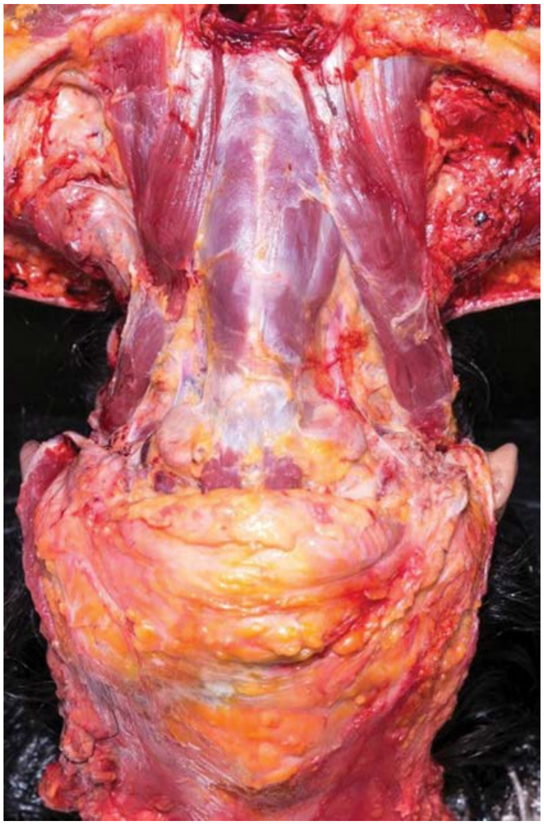

**Figure 10.10** Reflection of the skin and the platysma allows inspection of the superficial layer of the strap muscles. The sternocleidomastoid muscles can be seen along the sides of the neck and the paired sternohyoid muscles located centrally.

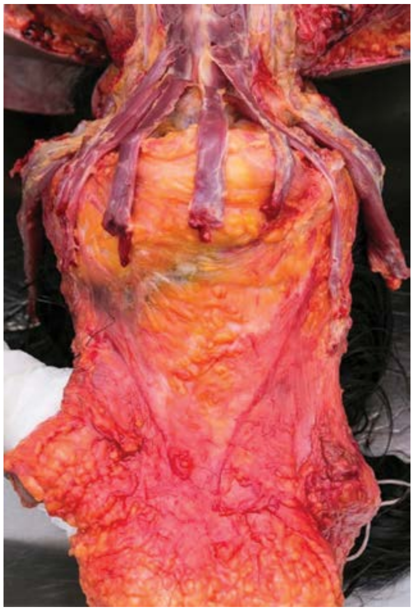

**Figure 10.11** Layered strap muscles after reflection of omohyoid, thyrohyoid, sternocleidomastoid, sternohyoid and sternohyoid muscles.

can be used to record injuries in the neck muscles. Example pre-drawn diagrams of the internal layers of the neck muscles are shown in Figure 10.15.

## Postmortem artefacts

There are five main artefacts/pitfalls in interpretation of postmortem findings [3]:

1. Developmental segments of the hyoid bone.
2. Anatomical variations including triticeous cartilages.
3. Prinsloo and Gordon haemorrhages.
4. Postmortem hypostatic haemorrhages.
5. Resuscitation-related neck injuries.

### Developmental segments of the hyoid bone

The hyoid bone is part of the hyoid–laryngeal complex which forms the internal hard structures of the throat. Discontinuities of the hyoid bone may be interpreted as fractures by an untrained person. The greater cornua and the body of the hyoid bone in early development stage are present as three separate bony structures. During the developmental progression, the joints are progressively ossified. Due to variable developmental progression, these bony structures may not be symmetrical in some individuals. In such cases, the synchondrodic joints may be unfused or partially unfused on one side of the hyoid bone. In the presence of artefactual haemorrhages, dysmobility can give rise to misinterpretation as fractures.

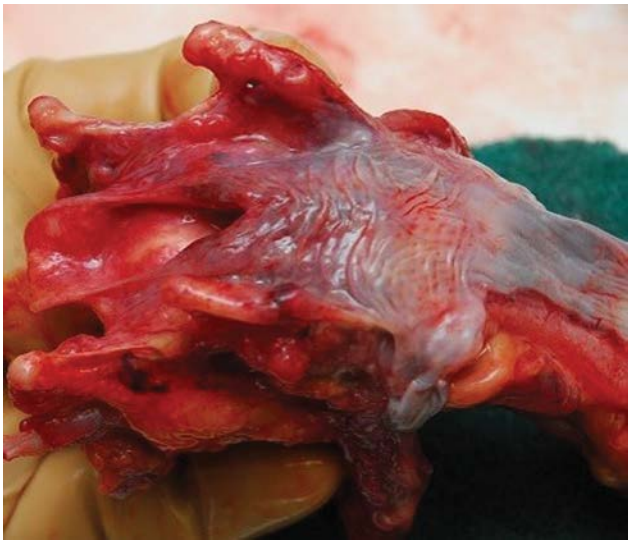

**Figure 10.12** Larynx after removal from the neck, showing hyoid bone and epiglottis on left side and superior horns of the thyroid cartilage. Note the fractured left superior horn (lower part of image) with associated hemorrhage.

### Presence of triticeous cartilages

Triticeous cartilages are small pieces of fibrocartilage which are linear or round and a few millimetres in size. They are

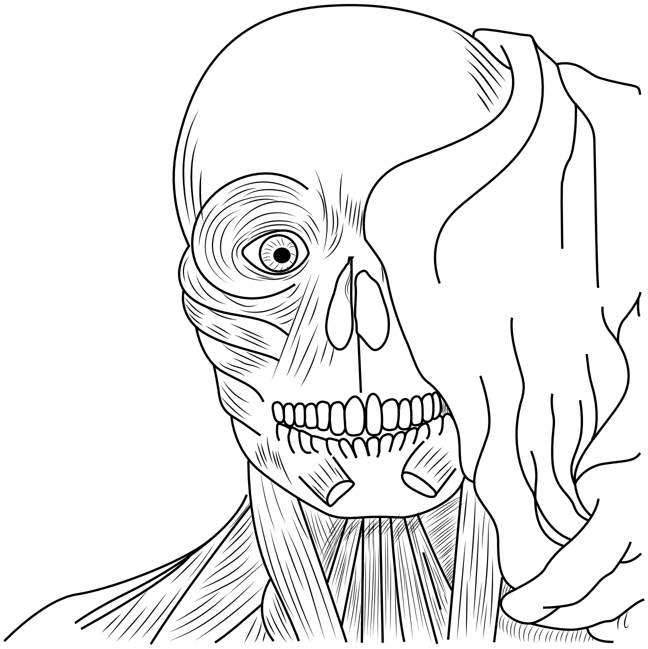

**Figure 10.13** A diagram showing a facial dissection.

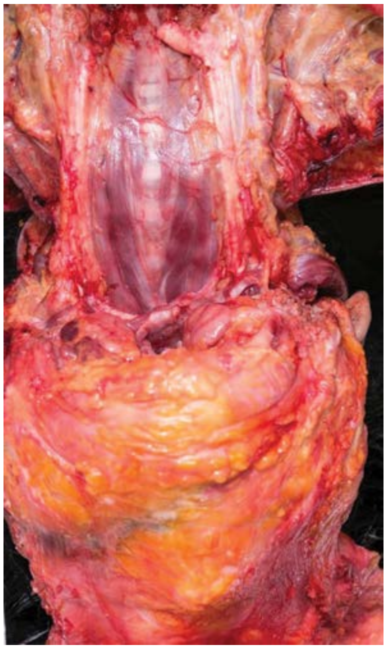

**Figure 10.14** The vertebral bodies and para-spinal muscles in the middle and common carotid arteries at the periphery after removal of the larynx.

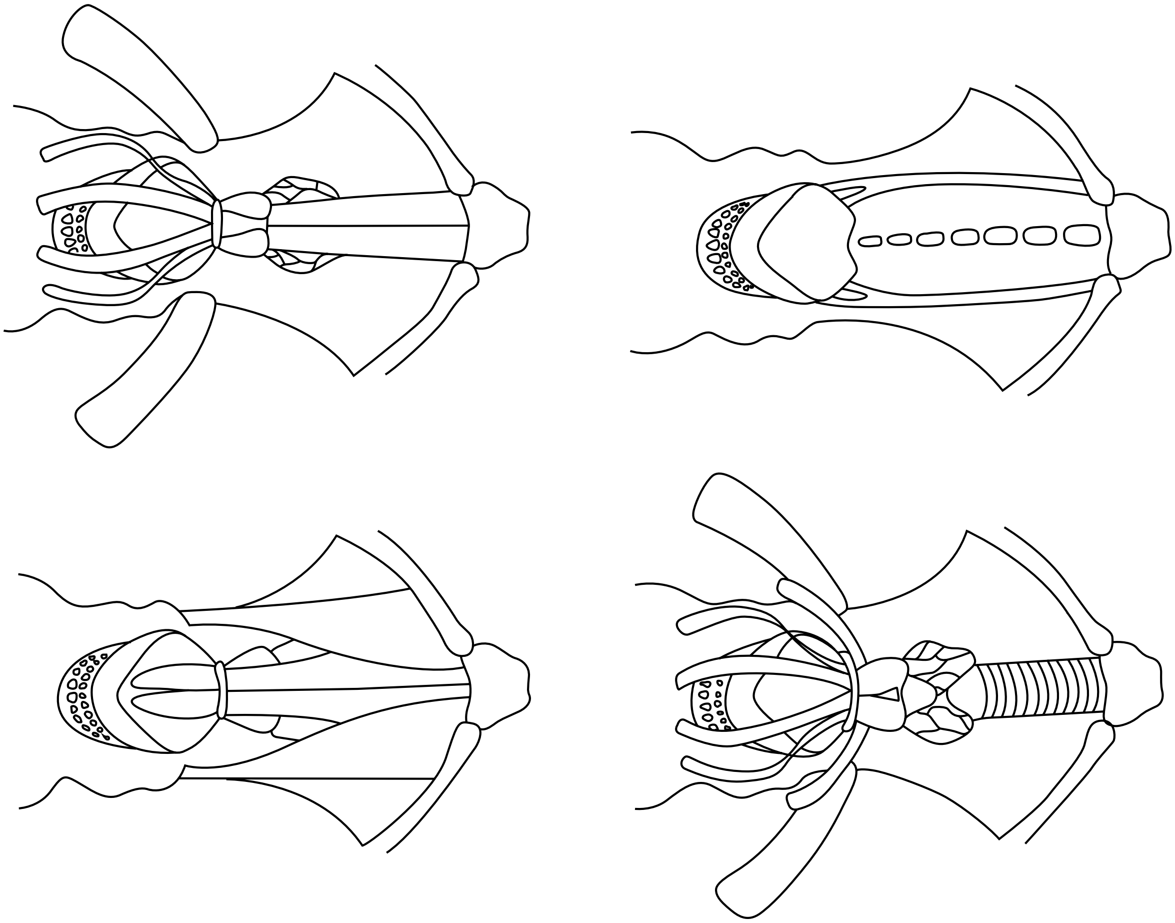

**Figure 10.15** Diagrams of the internal layers of the neck muscles that could be used to record injuries.

present in the fibrous connective tissue that links the superior horns of the thyroid cartilage to the suspensory ligaments and the soft tissues of the superior/anterior neck, adjacent to the hyoid bone. The presence of triticeous cartilage can be misinterpreted as a fracture of a bone in this location.

### Prinsloo and Gordon haemorrhages

These artefactual haemorrhages were named after the great South African forensic pathologists Prinsloo and Gordon [4]. They can lead to misinterpretation of extravasation of blood into the soft tissues of the neck as a mimic of bruising due to trauma. Postmortem handling of the neck structures such as blunt dissection and incisions made during the neck dissection are the true cause of these haemorrhages. The congested blood in this area can cause blood tracking between and within the strap muscles. This artefact can be avoided by dissection of the neck after vascular decompression and systemic layered dry neck dissection.

### Postmortem hypostatic haemorrhages

Postmortem hypostatic haemorrhages are due to extravasation of blood into the interstitial tissue. This occurs as a result of congestion of the venous plexus and distention of the venous plexus due to gravitational hypostasis. As a result of postmortem changes, the vascular integrity can be breached and the blood can be extravasated from the vessels into the soft tissues.

### Resuscitation-related neck artefacts

There are three main resuscitation-related neck injuries that can lead to interpretation difficulty.

1. Injury to sternomastoid muscle as a result of placement of a cannula in the internal jugular vein.
2. Laryngeal mucosal haemorrhage and oedema related to endotracheal intubation.
3. Laryngeal fracture as the result of cricoid pressure during intubation and resuscitation.

Another uncommon postmortem artefact is extravasation of blood within the strap muscles and the platysma in cases of drowning or due to immersion in water.

Blunt-force neck trauma also needs to be differentiated from injuries caused by neck compression. Often, direct impact to the neck without neck compression does not lead to florid petechial haemorrhages.

## Interpretation of errors

Interpretation errors can occur due to improper recording of data/findings, lack of knowledge of postmortem artefacts, lack of knowledge of anatomical variations and not following the established stepwise approach in systemic death investigation.

Awareness of the anatomical variations, postmortem artefacts and other pitfalls in the neck is essential. The neck represents a critically important anatomical structure in forensic medicine. Surface artefacts, such as a neck fold of an infant or an obese person, may be mistaken for a ligature mark. Injury caused by tight clothing around the neck, in particular when the body is decomposed, can also mimic a ligature mark.

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# 11 Neuropathology

*Wolfgang Keil and Claire Delbridge*

Most asphyxiation events lead to death during or immediately after trauma. Usually, in the short time span of events, only minor pathological anatomical findings can be caused.

In rare cases, the survival time can be a few minutes, several hours or even many days. Such constellations are referred to as 'late deaths'. Those affected have experienced an almost fatal $O_2$ deficit as a result of which they have become unconscious. Depending on the severity of the trauma, various brain syndromes can develop, which usually lead to brain death. In the neuropathological examination of such cases, both macroscopically and microscopically serious findings can be detected. It should be noted that during longer survival periods additional complications may develop, such as pneumonia with sepsis.

## Death in close temporal connection with the asphyxiation process

According to pathophysiological findings, every acute $O_2$ deficiency leading to cerebral death causes changes within a very short time, as a result of which morphologically detectable findings are formed. The brain always reacts with the development of a generalized oedema. Thus, it is not possible to draw conclusions about the actual cause of the $O_2$ deficiency by simply looking at the oedema. Acute hypoxic–ischaemic damage cannot be distinguished from purely hypoxic damage. The consequences of a disease-related drop in cerebral $O_2$ concentration, for example caused by coronary heart disease, therefore cannot be distinguished from traumatically caused $O_2$ deficiency. The severity of the findings caused by $O_2$ deficiency can vary considerably from case to case, even with the same cause of death. In individual cases, the oedema may be very mild or macroscopically imperceptible.

In this respect, forensic–neuropathological investigations in connection with cases of asphyxiation essentially have the aim of excluding pre-existing diseases of the central nervous system, or of obtaining indications of such diseases, which in individual cases could have significance for the assessment of the events.

Generalized cerebral oedema occurs within the few minutes from the beginning of the impairment or termination of the cerebral $O_2$ supply to the onset of death. This finding occurs in many cases of asphyxiation. Significant oedema may apparently develop if asphyxiation is accompanied by a severe obstruction of the venous drainage from the head, as can happen in ligature strangulation or pressure congestion according to Perthes. On the other hand, experience has shown that, in the case of deaths caused by soft covering of the mouth and nose as well as in aspirations with a dislocation of the trachea and bronchi, pronounced cerebral oedema is less common. However, there are no reliable connections between the type of asphyxiation and the intensity of the cerebral oedema.

Oedema develops as a result of the rapid drop in oxidative metabolism in the neurons, the glia, the endothelial cells of the blood vessels and the choroid plexus. The abrupt lack of energy leads to the breakdown of homeostatic conditions. In the acute hypoxic–ischaemic phase, the ion pump of the neuroglia and neurons, in particular the activity of the $Na^+/K^+$–ATPase, quickly comes to a standstill. As a result, the $Na^+$ concentration increases intracellularly and the $K^+$ concentration decreases. The resulting membrane depolarization leads to $Cl^-$ influx into the cells. Due to osmosis, water reaches the intracellular space and the cells swell. The glial cells in particular absorb water in order to compensate for the intracellular increase in osmolarity. Aquaporin-4 obviously plays a role as a mediator. This intracellular oedema, called cytotoxic oedema, occurs first. The subsequent collapse of the blood–brain barrier causes the formation of vasogenic oedema. As a result, proteins are transported extravasally and draw water into the interstitial space through osmosis. As a result, the volume of the interstitial space increases [2,4,5].

The severity of generalized cerebral oedema can now be reliably assessed by computer tomography. This is particularly important for the survival of a sudden cerebral $O_2$ deficiency (e.g. in patients after an attempted hanging). The severity of the developing oedema is a clinical indicator for the prognosis of the patient. As the pressure increases, the perfusion of the brain and thus also the $O_2$ supply is consecutively reduced. Consequently, secondary brain damage may occur [3,4].

In pathological anatomical findings, the severity of the generalized oedema is characterized by the increased weight and volume of the brain. The weight can be about 200 g, in extreme cases even about 300 g, above the normal brain weight of men and women. The presence of cerebral pressure signs in particular proves that there has been a considerable increase in pressure. Brain pressure signs are caused by the exhaustion of the remaining intracranial space, which forms only about 5 per cent of the total intracranial volume. If this spare volume is filled as a result of brain enlargement, an additional increase in cerebral pressure occurs, which reduces cerebral blood flow and ultimately leads to the loss of autoregulation of the cerebral blood vessels. This results in shifts in brain masses with entrapment of brain tissue, which can be detected during autopsy on the surface of the brain. As a sign of the infratentorial space requirement, the cerebellar tonsils are constricted by herniation into the foramen magnum. The constriction can take the form of a cone, which is then called a pressure cone. The morphological correlate of the supratentorial space requirement are grooves on both sides of the uncus, which result from herniation at the tentorium. Both findings may be present in isolation or in combination. In such cases, flattening of the gyri and narrowing of the sulci can be observed at the same time. Fresh cut surfaces are usually wet, soft and pale. The cerebral cortex, Ammon's horn, thalamus and striatum are particularly affected, while the basal ganglia are generally less affected. The border of the grey and white matter is often blurred. Blood vessel borders may appear blurred by the free fluid in the interstitium. The ventricle system is narrowed. All changes are largely symmetrical [1,6].

## ■ Late deaths

In cases of late death, the brain is macroscopically softened to liquified as a result of incipient or advanced autolysis. The walls and surrounding tissue of the lateral ventricles are often particularly affected by softening. Substantial cerebral autolysis may then contrast with the much smaller autolytic changes of the other organs. Often it is no longer possible to detect cerebral pressure signs in the softened brain.

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# 12 Neurohistology

*Wolfgang Keil and Claire Delbridge*

## Death in close relation to the asphyxiation process

As discussed in Chapter 11, many asphyxiation cases are dominated by generalized cerebral oedema, which can be diagnosed well according to macroscopic criteria. In contrast, its diagnosis in microscopic specimens is sometimes difficult because there is no histological staining for the direct representation of water retention. Oehmichen et al. [8] have pointed out the problem of the visualization of brain oedema. For example, a macroscopically detected cerebral oedema may occasionally not be recognizable by light microscopy. 'Empty' spaces, particularly around blood vessels, are indicative of an oedema. Frequently, only a discrete loosening of the tissue matrix can be observed, which can also have a spongy effect when more pronounced. The white matter is particularly affected as a result of fluid deposits in the interstitium. In the cerebral cortex, astrocytes essentially absorb water, especially in their perivascular processes [1].

Death as a result of an $O_2$ deficiency lasting for a few minutes can be explained functionally. Without $O_2$, the catabolic metabolic processes of cellular respiration cannot take place and a functional breakdown occurs. This period of time is so short that structurally visible damage under the light microscope can hardly develop.

For example, Kitamura [3] was able to detect no or only very slight neuronal damage on samples of hanging cases using light microscopy. If at all, brain structures that are particularly sensitive to hypoxia or ischaemia are primarily affected. These are the pyramidal cells of the CA1 region of the hippocampus, the neocortical layers and the Purkinje cells of the cerebellum. There, elective nerve cell destruction occurs more frequently. This is characterized by shrinkage of the neurons, which have condensed cell nuclei and a strongly eosinophilic cytoplasm. Shrinkage of the Nissl bodies may be visible. These findings are non-specific and do not justify conclusions to be drawn about the underlying event. A differentiation between the consequences of a pure hypoxia and a hypoxic–ischaemic trauma is also not possible by means of light microscopically recognizable neuronal changes. It should be noted that nerve cell shrinkage can also occur artificially, for instance due to insufficient fixation of the examination material [1].

Using immunohistochemical methods, isolated cellular damage has been detected. Kühn et al. [4] investigated asphyxiation cases with the neuron marker microtubule-associated protein 2 (MAP2). The proportion of MAP2-positive neurons was significantly reduced in these cases compared to a control group. In no case did a survival time exist. The phenomenon, which can be interpreted as an expression of a very rapid nerve cell change or damage, was found both in the hippocampus and in cerebral cortex layers. Kitamura [3] occasionally observed swelling and low proliferation of the cell bodies of GFAP-positive astrocytes (GFAP = glial fibrillary acidic protein) as early changes after traumatic $O_2$ deficiency. Occasionally, vimentin-positive astrocytes were also shown in the subpial region. Oehmichen et al. [8] pointed out that there was no method to detect acute hypoxic damage in the brain. At the same time, the authors drew attention to the fact that an $O_2$ deficit occurs during agony in the process of dying, which cannot be distinguished from the life-threatening acute hypoxic trauma.

## Late deaths

As early as 1960 Wünscher and Möbius reported the pronounced brain changes found light-microscopically in the case of an initially survived hanging [9]. The patient died after 2 days. Bronchopneumonia was ultimately regarded as the cause of death. Later, at least 2–5 hours were reported by Kitamura [3] and 7 hours by Oehmichen et al. [8] as the survival time for the occurrence of clear light microscopic findings. Initially, serious nerve cell damage develops, which is localized in almost all regions. At this point, glial cell reactions do not have to be present [3]. The nerve cells have pycnotic nuclei, and karyolysis may occur. The perikaryon becomes very strongly eosinophilic. The myelin sheaths decay, the oligodendroglia cells shrink. The astrocytes can show considerable cytoplasmic swelling. Astrocytes and oligodendroglia cells often show karyolysis also. After about 12 hours, segmented granulocytes emigrate. After about 24 hours, the proliferation of the microglia begins, which converts into macrophages after 48–60 hours at the latest. Activated microglial cells and macrophages are an expression of resorptive reaction. In particular, the released lipids from the disintegrating myelin sheaths are stored in cells, classified as lipid-laden macrophages. In the further course, erythrocytes or haemoglobin escaping vessels as a result of diapedesis can be phagocytized. These storage cells containing haemosiderin granules have been described as siderophages and can also be represented by a positive iron reaction. Lymphocytes occur more frequently. Later, astrocytes begin to proliferate and phagocyte part of the extracellular detritus together with the macrophages. During these still vital cellular reactions, death occurs in all cases because excessive oedema in the supratentorial parts of the brain occurs at the same time, which ultimately leads to the complete cessation of existing intracranial perfusion. As a result, the brain softens and decays autolytically.

After prolonged survival, the cellular response may be absent in rare cases. This can occur if the asphyxiation process has led to a severe or even complete interruption of the cerebral blood flow for a few minutes. A reperfusion after this trauma is then no longer possible, due to the immediately developing oedema with rising cerebral pressure. The cerebral pressure can rise higher than the systolic blood pressure in a short period of time. This leads to a total ischaemic infarction of the brain. Even if the affected person receives intensive medical care for hours or even days until the diagnosis of brain death has been completed, physiological cellular reactions in the brain are no longer possible. Microscopically, necrosis of not only the nerve cells but also of all other cells is found. The release of lysosomal enzymes leads to the general autolysis of all brain structures. Such an event was also referred to as ‘intravital autolysis’. In normothermal conditions, an extensive ‘intravital brain autolysis’ manifests within 24–48 hours. The brain becomes generally soft. Necrotic pressure cones develop at the cerebellar tonsils. The autolytic brain can show a demarcation of the pituitary gland, optic nerve and medulla oblongata [1].

The cerebral changes which develop after survived asphyxiation have been investigated several times with immunohistochemical methods. Data on survival times of only 30 minutes are available from Li et al. [5]. There, certain differences in the astrocytes between patients with different causes of death were described. The number of positive astrocytes for GFAP and S100B protein in the hippocampus was comparatively reduced in strangulations, such as atypical hanging. Such reduction was not observed in other types of asphyxiation, such as aspiration, but also not in samples of cases after acute myocardial infarction. The reduction in the number of S100B-positive hippocampal astrocytes in the strangulation cases was described to be accompanied by an increase in S100B serum levels.

Axonal damage after hypoxic–ischaemic attacks was observed in high frequency after a survival time of at least 3 hours, especially in the pons, but also in other brain regions [6,7]. The substrate is expressed β-amyloid precursor protein (β-APP). However, these findings are unspecific with regard to the actual mechanism of death. Axon damage can be observed not only after hypoxic–ischaemic alterations, but also after craniocerebral trauma due to blunt violent effects and in cases of multiple sclerosis. Axon lesions are also discussed as secondary damage. They could possibly result from brain oedema [7].

**Table 12.1 Neuropathologically relevant indicators of previous cerebral $O_2$ restriction**

| Marker | | Relevance |
|---|---|---|
| MAP2 | Microtubule-associated protein 2 | As a neuronal marker |
| ALZ 50 | Tau protein in Alzheimer’s disease | Also microtubule-associated protein |
| GFAP | Glial fibrillary acidic protein | As a glial marker |
| CD68 | Cluster of Differentiation 68 | As a marker for microglia and macrophages |
| β-APP | β-amyloid precursor protein | To visualize axonal damage |

It is generally assumed today that changes in the neurons are detectable about 7 hours after a relevant cerebral $O_2$ deficit [3,8]. Oehmichen et al. [8] have presented a selection of neuropathologically relevant and applicable markers, which may indicate a previous restriction of cerebral $O_2$ supply (Table 12.1)

An assignment of the findings to certain types of asphyxiation is not possible on the basis of the marker reactions (e.g. a distinction as to whether the neuronal damage was caused by strangulation or by drowning).

Oehmichen et al. [8] have dealt comprehensively with posthypoxic reactions of the brain. First, nerve cell destruction becomes visible in all brain regions. Astrocytes accumulate and proliferate, and neutrophil granulocytes and macrophages migrate, ingesting the apoptotic cell material. Within 12–24 hours the number of activated microglia and macrophages increases considerably. Finally, in the late phase after hypoxic or hypoxic–ischaemic lesions, an increase in activated astrocytes occurs with an increase in GFAP positivity. The authors point out that the reactions are unspecific.

Kitamura [3] also found that the detection of hsp70 (hsp70 = heat shock protein70) in nerve cells in connection with proliferating GFAP-positive cells may indicate prior transient severe $O_2$ deficiency in affected deceased.

Immunohistochemical investigations on the brains of deceased newborns and infants were published by Fineschi et al. [2]. The aim of the studies was to estimate the time of onset of perinatal hypoxic–ischaemic cerebral $O_2$ deficiency. For this purpose, hypoxia-induced markers such as heat-shock proteins (hsps) and osteogenic regulatory protein (ORP150) were stained in addition to the commonly known inflammatory markers. Depending on the pattern of immunohistochemically positive findings, it was possible to make rough estimates of the time at which the brain damage developed. Clinical questions of this kind can also become of forensic significance. Furthermore, the authors have successfully used the pattern of other markers present in the serum and cerebrospinal fluid (e.g. S100B and LDH) to assess the prognosis after perinatal hypoxic–ischaemic brain damage.

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# 13 Histopathology of the Lung in Asphyxiation, Suffocation and Pressure to the Neck

*Wolfgang Grellner and Burkhard Madea*

## Introduction

In the medicolegal investigation of fatal cases of asphyxiation, marked external signs of vitality such as cyanosis/congestion or petechiae may be missing. The time course or the survival time is frequently of considerable relevance. The problem of the correct diagnosis of vital asphyxiation is aggravated as meanwhile most of the so-called ‘vital reactions’, in particular in cases of hanging, are drawn into doubt. For these reasons the histological and immunohistochemical examination of pulmonary tissue has been evaluated by several authors.

As a central organ in the pathophysiology of asphyxiation, the lung may exhibit alterations of its microstructure and/or cell content. In this context the working group of Brinkmann et al. [3,4] established the diagnosis of asphyxiation by microscopic investigation of the lung: they emphasized characteristic pulmonary alterations such as emphysema, alveolar–septal oedema, hyperaemia and microhaemorrhages, which they named ‘haemorrhagic–dysoric syndrome’ due to enhanced permeability of membranes. In combination with microembolism syndrome, they regarded it as a tool to differentiate death by obstructive asphyxia from other causes of death with similarly short periods of agony. Janssen, in addition, observed the appearance of numerous alveolar macrophages and intra-alveolar giant cells in cases of protracted oxygen deficiency (throttling, smothering, thoracic compression), an agonal mobilization and proliferation of alveolar cells with the formation of multinuclear giant cells [9,10]. In contrast, Betz et al. [1,2] demonstrated that these cell populations appeared in both fatal asphyxia/suffocation (strangulation, drowning, thoracic compression, hypoxia) and control cases with very short survival periods with nearly the same frequency.

## Methods

For studying routine lung histopathology, at least one sample of each lobe should be taken during autopsy. After fixation in buffered formalin and paraffin embedding, standard methodology should include staining with HE, PAS and naphthol-AS-D-Cl-acetate-esterase and, if necessary, Pappenheim for better imaging of cell subtypes. In addition, frozen sections of fixed tissue should be prepared for the detection of fat components. The microscopic evaluation of cases follows with regard to general morphological changes (alveolar and interstitial oedema, hyperaemia, alveolar and interstitial haemorrhages, emphysema, dystelectasis) and especially alterations of lung blood vessel contents. The former can be graduated in a semi-quantitative way (<10%, 10%–50%, >50%; or absent, discrete, strong/severe), the latter divided into fat embolism (degree I–III), bone marrow tissue embolism (count) and intravascular cell accumulations. These can be regarded as positive, if ≥20 per cent of the cells in a vessel are non-erythrocytic. The percentage of positive vessels is then noted and graduated (<5%, 5%–20%, 21%–50%, >50%).

For special purposes, immunohistochemical techniques for the detection and characterization of alveolar macrophages and pulmonary giant cells may be performed. A panel of selected markers has been proposed as follows:

- CD68, LN–4, etc.: general markers of macrophages.
- 27E10: early-stage inflammation marker.
- 25F9: late-stage inflammation marker.
- AMH152: marker of activated macrophages.
- Ki-67: proliferation marker.

A semi-quantitative count of positive cells seems to be sufficient (negative, weak, moderate, strong reactions; number of cells per microscopic field).

## Main results and meaning

In accordance with macroscopic findings, general structural changes can be observed in all types of strangulations more or less frequently. They include interstitial and intra-alveolar oedema, associated hyperaemia and alveolar haemorrhages and, moreover, focal emphysema and local dystelectasis (Figures 13.1 and 13.2). Apart from emphysema (present in about 10%–40% of cases) the

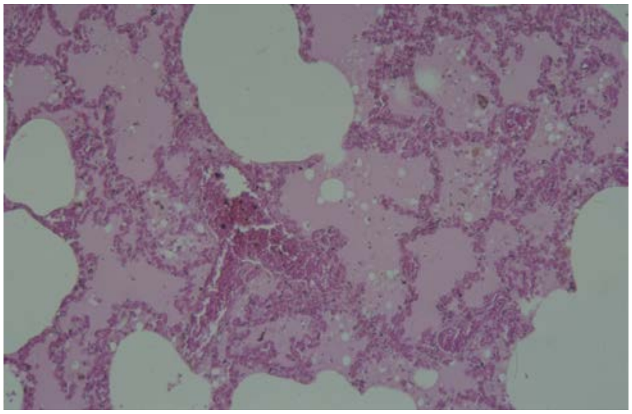

**Figure 13.1** Oedema and hyperaemia of the lung, HE (×100).

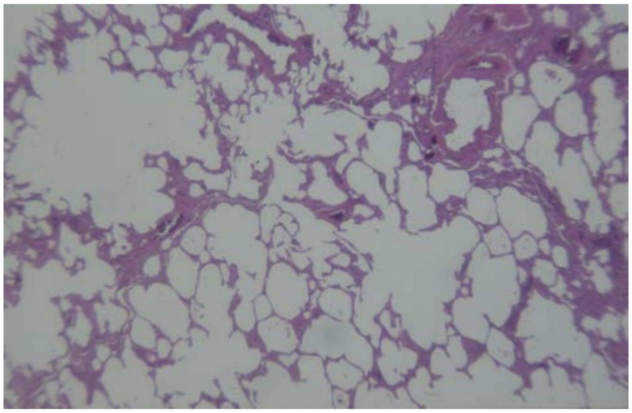

**Figure 13.2** Emphysema of the lung, HE (×50).

phenomena appear quite regularly (frequencies in the range of mainly 70%–100%). Significant differences between the groups (typical and atypical hanging, ligature strangulation, throttling, further combinations) cannot be distinguished. Oedema can be found in nearly 100 per cent of cases (among these in one-quarter severe oedema involving more than 50% of alveoli). In a minority of cases with beginning intra-alveolar oedema, perivascular and interstitial oedema can be distinctly differentiated. In about one-third of fatal strangulations, small air bubbles within the alveolar oedema fluid are present. Strong hyperaemia is a very regular finding and is nearly as frequent as oedema. Alveolar and interstitial haemorrhages also occur frequently, but mainly in a very discrete manner. Comparison with control groups (e.g. sudden cardiovascular death) and the general experience, however, demonstrates that the complex consisting of oedema, hyperaemia, haemorrhages and dystelectasis must be regarded as non-specific for a strangulating event. Focal emphysema does not appear in control groups (without resuscitation measures), is not so frequent in strangulations and within the single case mostly not very extensive. On the whole, these general structural changes of the lung are non-specific for asphyxia/strangulation/suffocation, also present in control cases, and there is no evidence of so-called 'haemorrhagic–dysoric syndrome'.

Microscopic alterations of the pulmonary blood vessel contents consist of three main phenomena: fat embolism, bone marrow tissue embolism and intravascular cell accumulations.

Fat embolism appears in only a minority of cases and regularly to a minor degree. As with bone marrow tissue embolism (similarly low frequency), it is nearly always restricted to cases with accompanying violence such as fractures and blunt trauma, or cases with resuscitation measures. Both phenomena are rarely observed. In particular, bone marrow tissue embolism occurs mainly sporadically (once or twice a case). Both phenomena can be seen more often in control cases.

Intravascular cell accumulations consist of polymorphonuclear and juvenile granulocytes, lymphocytes, some monocytes and obviously immature bone marrow cells (Figure 13.3). About one-half to two-thirds of cells give positive stains with naphthol-AS-D-Cl-acetate-esterase and belong to the myeloic system. The cell accumulations concentrate on medium-sized and smaller arteries and appear partly as aggregates filling the whole vessel and partly in a disseminated manner. On the whole, these changes are mostly restricted to a few sections of a case or even to a few vessels within one section. Most frequently, less than 5 per cent or certainly less than 20 per cent of all vessels show positive reactions in this sense. The phenomenon is more frequent in non-hanging cases (approximately one-third vs one-tenth). In all groups it is mainly limited either to protracted agony courses or to cases with accompanying blunt force (including antecedent resuscitation measures). The phenomenon of intravascular cell accumulation is present in control cases as well when (longer) resuscitation is documented/probable.

On the whole, it is therefore mandatory to take the criteria 'accompanying force' and 'possible resuscitation measures' into special consideration in the evaluation of lung histology in strangulations.

On the basis of our own experiments and experience (Table 13.1) and with the background of the results reported in the literature, the following conclusions are drawn [6]:

- The regularly observed general changes of the lung structure (e.g. oedema, hyperaemia) are undoubtedly non-specific for strangulations/asphyxiation.
- The alterations of the blood vessel contents may serve as a general vitality marker, if resuscitation measures are excluded, but not as evidence of strangulating force. In cases without signs of blunt force, they point to protracted agony courses in the sense of shock equivalents.

The occurrence of numerous alveolar macrophages and pulmonary giant cells has been reported in both fatal asphyxia and other causes of death. There is controversial discussion on the issue of whether, in particular, the

**Table 13.1 Selected case histories with special findings**

| | | | | | | Microscopic lung structure | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| **Case** | **Age (years) and sex** | **Case group** | **Presumable survival period** | **Resus-citation** | **Accompanying blunt force** | **Oedema** | **Fat embolism** | **Bone marrow tissue embolism** | **Intravascular cell accumulations** | **Particularities** |
| 1 | 24<br>Female | Atypical hanging | Minutes | ? | – | +++ | – | – | (+) | Hanging in kneeling position |
| 2 | 53<br>Female | Ligature strangulation | Minutes–1 h | – | +++ | +++ | ++ | + | ++ | None |
| 3 | 25<br>Female | Ligature and manual strangulation | Several h | ? | ++ | + | – | – | ++ | Numerous alveolar giant cells |
| 4 | 20<br>Female | Ligature and manual strangulation | 30 min | – | (+) | +++ | – | – | + | Focal emphysema |
| 5 | 8<br>Female | Other compressing force against neck | Longer | + | +++ (cofactor for death) | + | (+) | – | +++ | None |
| 6 | 59<br>Male | Control group | Short, but resuscitation | 70 min | + (resuscitation) | ++ | + | – | ++ | Severe cardiac hypertrophy |

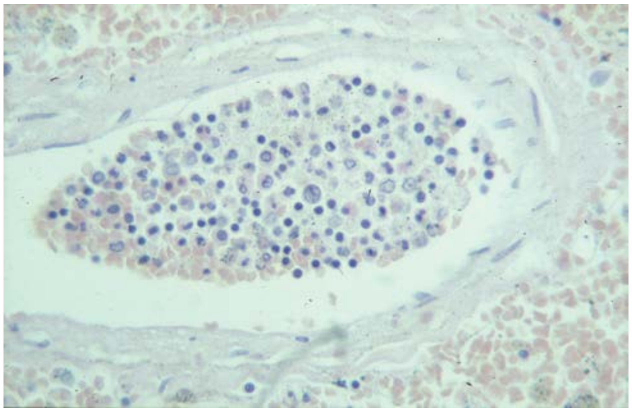

**Figure 13.3** Intravascular cell accumulation in lung tissue, HE (×500).

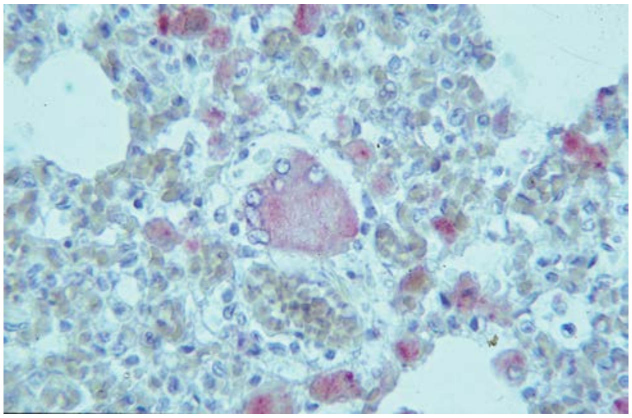

**Figure 13.4** Pulmonary macrophages and detail of giant cell, general marker CD68 (×500).

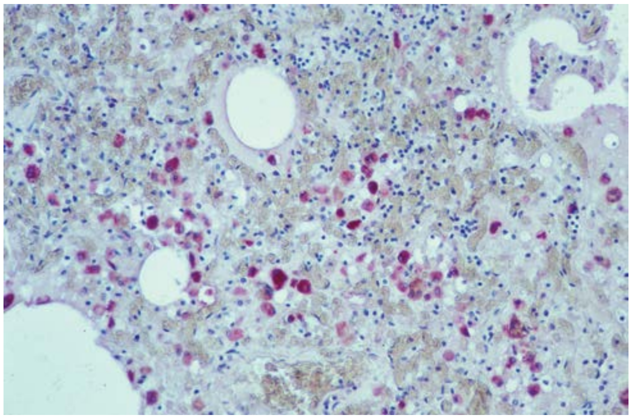

**Figure 13.5** Pulmonary macrophages, late-stage inflammation marker 25F9 (×200).

appearance and number of pulmonary giant cells can support the diagnosis and type of fatal asphyxia, for example with regard to the survival time (rapid or protracted asphyxia). The German forensic pathologist Janssen described a mobilization and proliferation of alveolar cells with the formation of multinuclear giant cells in fatalities with protracted oxygen deficiency [9,10]. By contrast, other authors such as Betz et al. [1,2] demonstrated that these cell populations appeared in both fatal asphyxia/suffocation and control cases (cardiovascular deaths) with nearly the same frequency. Further investigations including our own results [5,7,8,11] lead to different conclusions as to the role of pulmonary macrophages in asphyxia/suffocation. These cell types are not restricted to asphyxia. Their appearance is not of pathognomonic value for this diagnosis. Giant cells occurred even more than twice as frequently in opiate-involved deaths (group with prefinal oxygen lack) compared to strangulations. Both cell types seem to be of heterogeneous nature reflecting different functional states as only some general markers of macrophages are expressed by all cells (Figure 13.4). Positive reactions of alveolar macrophages or giant cells with markers of early-stage inflammation, activation or proliferation could not be observed. Varying results emerged with the late stage inflammation marker 25F9, approximately 70 per cent of alveolar macrophages and 20 per cent of pulmonary giant cells showed positive reactions in fatal strangulations (Figure 13.5); the staining pattern in other cases (opiate-involved deaths, cardiovascular deaths) was in a similar dimension.

These results do not suggest prefinal immigration, mobilization or proliferation of alveolar cells, but rather point to a longer pre-existence of macrophages and giant cells in pulmonary tissue. At present, a classification of these phenomena as vital parameters for asphyxia/suffocation or as a tool for differentiation between short and longer agony courses is not possible.

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# 14 Asphyxia-triggered Inflammatory Reaction Patterns of the Lung

*Ewgenija Gutjahr and Burkhard Madea*

## Introduction

The medicolegal diagnosis of a fatal asphyxia, especially in the context of the vitality of lesions, still remains a challenging issue due to the obligatory proof of the suffocating process or its marks on the corpse (e.g. ligature marks, periligature injuries, perioral and perinasal skin erosions). Despite the existence of certain unspecific 'classical macroscopical signs' (such as cyanosis, petechiae, liquid corpse blood, bleeding in the neck muscles or fracture of laryngopharyngeal skeleton), the identification of suffocation still requires the overall assessment of circumstantial data and morphological findings. This frequently involves the method of exclusion (e.g. after extensive toxicological analysis) in case of the lack of all these hints (e.g. due to a very rapid course of suffocation, the multimorbidity of the victim or the physical priority of the perpetrator compared to the victim).

For these reasons, there have recently been many attempts to identify pathomorphological criteria which are absent under physiological conditions and appear specifically in the context of asphyxiation, increasing in their expression with the duration of the hypoxic agony [8]. Thus, the focus of the research has been on the lung, a primary effector organ of the dysregulation of respiration and circulation due to a fatal suffocation.

Acute alveolar hypoxia, characterizing suffocation, is a physical state observed in various clinical situations and diseases, including anaphylactic shock, brain injury, intoxication and acute cardiac insufficiency. Clinical research therefore provides significant insight into physiological mechanisms, cellular changes and further asphyxiation markers in the lung, all of which are also helpful in forensic pathology.

## Microstructure of the lung and the role of alveolar macrophages and giant cells

Pathophysiologically, suffocation is characterized by reduced alveolar $pO_2$, which induces vasoconstriction of the pulmonary arteries in the lung periphery and a redistribution of blood from basal to apical lung segments in accordance with the Euler–Liljestrand mechanism. As oxygen is the main requirement for the secretion of necessary vasoactive substances, a mild lung injury results in inflammatory changes due to acute hypoxia [27]. Consequently, the permeability of blood vessels, generated by a high number of pores and vacuoles in endothelial cells, is increased, followed by increasing pulmonary extravasation of fluid, electrolytes and albumin. This has been shown by Stelzner et al. [35] in rat lungs after 48 hours of exposure to hypobaric hypoxia, as well as by Madjdpour et al. [27] after 1 hour of exposure to 10 per cent $O_2$. A macroscopic equivalence for these changes on the cellular level, interstitial and alveolar lung oedema, showing a foamy microscopic picture of a mixture of an eosinophilic exudate and small air bubbles formed during the terminal disturbance of the air flow, is observed clinically (Figure 14.1).

Taking up these issues, Brinkmann et al. [4,7] postulated a complex pattern of emphysema, microembolism syndrome and haemorrhagic–dysoric syndrome (standing for the combination of the extravasation of erythrocytes and an increased permeability of membranes) as pathognomic of obstructive asphyxia in their study of five primarily unexplained homicides in the 1980s. However, their comparative study in 50 rats and 15 rabbits of strangulation vs various alternative causes of death (e.g. overdose of anaesthetics) resulted in a relativization of the initial thesis by abolishing the exclusive value of these morphological findings. This study merely underlined the higher frequency and the higher degree of the lung alterations described above in the cases of fatal asphyxia compared to the controls. Although this hypothesis was widely supported by many experts, similar reaction patterns were soon also registered in sudden cardiovascular fatalities as well as in cases of drug overdosage, proving the lack of specifity. In our recent study of 28 cases of suffocation, 11 cases of sudden cardiovascular fatalities and 13 cases of traumatic deaths, we could refute Brinkman's theory of the specifity of 'haemorrhagic–dysoric syndrome' by observing dystelectatis, subpleurally accentuated emphysema, haemorrhagia and the presence of lung oedema in around 50 per cent of all investigated collectives [19]. Despite the non-specificity of the lung oedema per se, our results prove a correlation between suffocation and a higher severity of oedema, compared

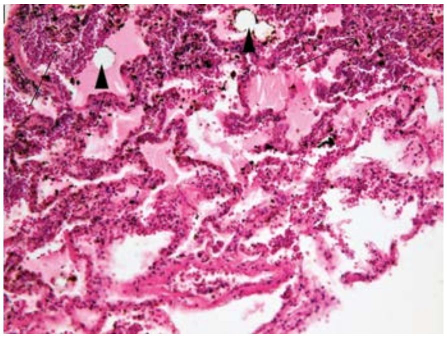


**Figure 14.1** A foamy histological picture of the parenchyma of a suffocated lung with lung oedema, intra-alveolar air bubbles (▶) and focal extravasation of erythrocytes as a sign of acute congestion (→) (×200).

to control cases: three-quarters of all suffocation deaths examined showed a strong lung oedema, whereas this severity of oedema was observed in only approximately 11 per cent of control cases (Table 14.1). In this context we also showed a shift of the frequency distribution of higher lung weights and of the higher degree of lung oedema to longer postmortem intervals. Despite a small collective, this tendency questions the representability of the severity of lung oedema for suffocation, implicating a possible artificial occurrence during hypostatic postmortal redistribution of body fluids. Apart from this ambivalent aspect, our findings are consistent with the results of the study of Grellner and Madea [17], performed on lung specimens of 106 fatal strangulations and 10 controls of sudden cardiovascular deaths, in which the authors evaluate the lung oedema as well as the microembolism as a further unspecific shock equivalent due to a prolonged agony course.

When analysing the primary initiator of the inflammatory reaction, triggered by hypoxia in the lung parenchyma, the key effector cells seem to be alveolar macrophages, localized at the air–tissue interface and therefore the first cell line in contact with inhaled substances. Following this theory, Janssen [22] and Janssen and Bärtschi [23] drew attention to the adaptive reaction of alveolar macrophages to hypoxia in the 1960s. In a study of four young victims of a prolonged asphyxia, followed by an animal experiment on 40 rats and guinea pigs whose death was caused by an interruption of oxygen supply of 30 minutes to 12 hours, they postulated a mobilization, a significant increase of the population of alveolar macrophages and their transformation to polynuclear giant cells with two to four nuclei, growing with the duration of preterminal asphyxia. They described an initial lining of the alveoli by pulmonary macrophages after a hypoxic period of 30–90 minutes. This was followed by the formation of morula-like intra-alveolar aggregates of macrophages, varying in their size, and a more frequent occurrence of giant cells as a result of the endomitosis of alveolar macrophages, responding to a protracted asphyxia, even as a supravital reaction after the individual's death.

Later attempts were made to elucidate Janssen's hypothesis of polynuclear giant cells as a useful specific indicator for slow asphyxiation. Betz et al. [2] contrarily showed the presence of polynuclear giant cells (defined as alveolar macrophages with at least three nuclei) in lung specimens of all groups in their study of 54 individuals with different natural and unnatural causes of death, except in cases of hypoxia due to covering the head with a plastic bag. Ranging from 22 per cent to 30 per cent of all pulmonary cells, no apparent differences between the groups, in particular any increased number in cases of throttling or strangulation, could be observed. In addition, the immunohistochemical determination of the proliferation activity of these giant cells did not show any significant increase of the Ki-67-index, thus indicating that these cells had not developed shortly before death by endomitosis as an adaptive change, responding to a reduced oxygen supply, as postulated by Janssen. The low Ki-67-index of alveolar macrophages was also verified by Grellner and Madea in their examinations of human asphyxiated lungs [17]. Although considering the elevated number of alveolar macrophages a diagnostic tool to differentiate a slow asphyxia from an acute one, Vacchiano et al. [38] also confirmed the lack of differences in morphometrically detectable numbers of polynuclear giant cells between a short (10–15 minutes) and a long (>30 minutes) asphyxia in their analysis of 50 asphyxiated human lungs. Grellner and Madea [18], moreover, found that there were more than twice the number of giant cells in the pulmonary tissue of opiate-involved deaths compared to that of the victims of throttling, thus declaring the connection between the occurrence of giant cells and hypoxia to be a random phenomenon.

**Table 14.1 Distribution of different degrees of lung oedema in asphyxia and two control groups**

| | | Lung oedema | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | Beginning | | Intermediate | | Strong | | Total |
| | | *N* | (%) | *N* | (%) | *N* | (%) | *N* |
| Cause of death | Suffocation | 6 | 60.0 | 17 | 58.6 | 7 | 77.8 | **30** |
| | Controls – traumatic deaths | 3 | 30.0 | 3 | 10.3 | 1 | 11.1 | **7** |
| | Controls – cardiovascular fatalities | 1 | 10.0 | 9 | 31.0 | 1 | 11.1 | **11** |
| | **Total** | **10** | **20.8** | **29** | **60.4** | **9** | **18.8** | **48** |

*Source:* From Gutjahr E, Madea B. *Forensic Sci Int* 2019;**297:**315–325.

Finally, deviating from Janssen's endomitosis theory, some contemporary authors who have detected a significant increase of giant cells and a clearly elevated number of immunohistochemically CD68-positive alveolar macrophages in suffocation deaths have suggested an alternative explanation of this phenomenon: the activation of the bone marrow and the immigration of inflammatory precursor cells from the bone marrow to the lung. Brinkmann declared the bone marrow embolism to be a sign of bone marrow activation. However, in our study [19] we observed this phenomenon in one control case and one death caused by choking. Due to the resuscitation performed, these changes seemed to be attributable to a concomitant blunt force with possible microfractures of the ribs.

Strunk et al. [36] regarded an increase in granulocytes and megakaryocytes in the parenchyma of asphyxiated lungs to be the histological equivalent to this hypoxic activation of the bone marrow. In their study of human lung specimens from the victims of a long ($n = 6$) and a short ($n = 8$) preterminal asphyxiation with trauma as controls ($n = 9$), they reported a four- to fivefold increase in the average number of megakaryocytes as well as a doubling or tripling of the pulmonary cell population of alveolar macrophages and giant cells in cases of protracted asphyxia.

Conversely, in our recent study [19] we detected low numbers of primarily intraseptally located megakaryocytes without a significant hypoxia-dependent elevation in all case groups (Figure 14.2c). We found that acute and chronic hypoxia can lead to an acceleration of proliferation and differentiation of bone marrow megakaryocytes and an increase of their functional activity. However, even in animal experiments on rats, this tendency was observed after as little as six hours [25], which must be considered in the context of the faster metabolism in rodents and the problem of transfer of experimental data to human cases under forensic conditions.

The accent on the duration of preterminal asphyxiation in various experimental systems may explain the differences in results. In our study [19] we designed the collectives of suffocation deaths according to most frequent forensic conditions. As a short period of agony, we took a classic course of events in, for example, typical and atypical hanging. After a total occlusion of airways by an extrusion tool, the exhaustion of oxygen reserves is reached at rest after 5 minutes, and in conditions of panic or struggle with the perpetrator in as little as 1 minute [16], [ causing a terminal collapse of blood circulation after 5–10 minutes. A prolonged asphyxia, found in choking and throttling, for example, was assumed in cases of partial occlusion of airways or incompletely prevented respiratory excursions, enabling an inhalation of small amounts of oxygen, thus prolonging the survival to 12–15 minutes. Using autopsy reports, a prolonged asphyxia was assumed considering the overall picture of concomitant injuries (as a sign of a

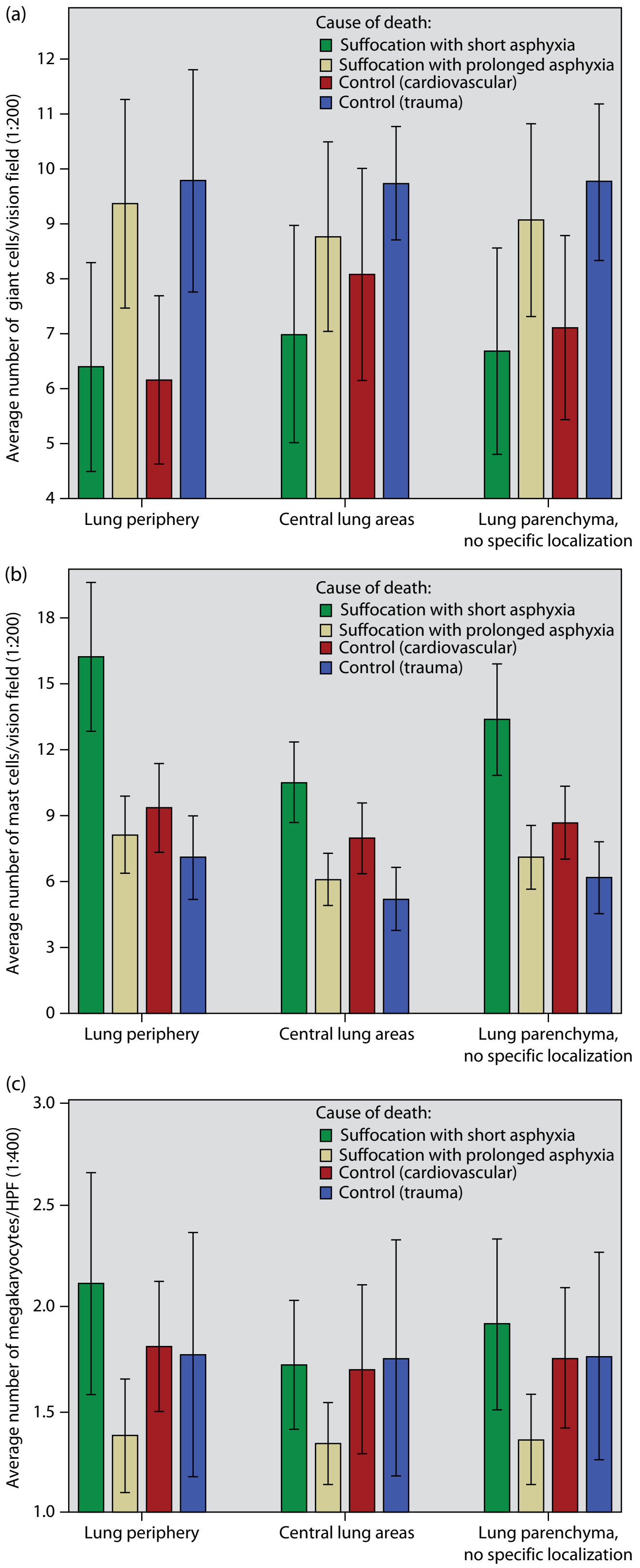


**Figure 14.2** Results of counting cells in subpleural and central lung areas. (a) Giant cells (cells/VF (×200)); (b) mast cells (cells/VF (×200)); (c) megakaryocytes (cells/10 HPF) (From Gutjahr E, Madea B. *Forensic Sci Int* 2019;**297:**315–325). VF = vision field; HPF = high-power field (×400).

defence of the victim and a longer survival) as well as the severity of cyanosis, congestion and petechiae, implicating a longer preterminal maintenance of a minimal blood circulation. A similar configuration of suffocation groups was used by Grellner and Madea [17], resulting in similar cell counts. In contrast, the animal experiments of Janssen and Bärtschi [23] as well as suffocation deaths chosen by most investigators [36] featured a duration of asphyxiation of around 30–90 minutes. A similarly long duration is not normally described for forensic cases of human suffocation deaths, except in rare cases of especially cruel behaviour by the perpetrator.

Nevertheless, the morphometric analysis of six vision fields (three subpleural and three central areas of each lung specimen; ×200 magnification) of each of five lung lobes per case detected a doubling of the giant cell number in the suffocation collective with a short agony ($n = 13$) and no significant differences in cases of a long asphyxia ($n = 15$) in comparison to the controls (sudden cardiovascular fatalities ($n = 11$), traumatic deaths ($n = 7$)) in our study [19] (Figure 14.2a). However, we consider these changes to be an expression of the individual variability in giant cell counts in healthy and functionally normal lungs. It is conceivable that various individual stimuli such as smoking, air pollution, etc., can induce the formation and regional accumulation of giant cells, particularly with regard to the mean lifespan of these cells of several days [11].

Similar research findings were presented by Betz et al. [1] in their study of 13 cases of homicidal strangulation or throttling, 8 cases of traumatic asphyxia due to chest compression and 10 controls. They observed a considerable variation in all groups investigated, with a tendency to higher numbers of cells in smokers or older individuals, showing no significant differences between inflammatory cell populations of the lung parenchyma in asphyxia and controls.

The assumption of interindividual variation of the inflammatory cell populations of the lung is further supported by the lack of noteworthy differences in giant cell numbers in cases of a short vs long asphyxia as well as by the observed accumulation of giant cells, alveolar macrophages, mast cells and megakaryocytes in subpleural areas compared to central parts of the lung in our study [19] (Figure 14.2a–c). This intraparenchymal cell distribution corresponds to the physiologically pre-existing gradient of the inflammatory cells in the lung, which can be explained by the hypostatically caused accumulation of all inhaled pathogens and foreign particles in peripheral lung sections with perfusion–ventilation mismatch (e.g. the principle of the development of pleura anthracosis).

Although our findings concerning the cell counts of giant cells contradict many other authors, it must be emphasized that even a hypoxia-induced formation of giant cells described for many types of cancer (e.g. sarcoma) in the literature requires an oxygen depletion of at least 7 days, as experimentally shown for the formation of osteoclasts (bone-specific giant cells) in feline cell culture [29].

In summary, the recent controversial data from the literature appears to support the recruitment of residential inflammatory cells of the lung as well as their release of high amounts of pro-inflammatory cytokines and chemokines as a response to the first minutes of asphyxia rather than an increase of the pulmonary number of inflammatory cells by an immigration from the hypoxically activated bone marrow or by proliferation and endomitosis.

Finally, the following practical conclusions for the morphological diagnosis of asphyxiation by evaluating microstructural changes of the lung and by detection of polynuclear giant cells and alveolar macrophages can be drawn:

1. The alterations of lung parenchyma (e.g. haemorrhagic oedema, subpleural emphysema, local dystelectasis and microembolism) consolidating Brinkmann's haemorrhagic–dysoric syndrome can be observed non-specifically in cases of various causes of death. However, the oedema in suffocated lungs features a higher degree of expression compared to control collectives. To discriminate intravital oedema from postmortal hypostatic fluid extravasation, a long postmortal period ante sectionem is to be excluded.
2. Polynuclear giant cells can also occur in the lungs of healthy, non-asphyxiated individuals and considerable variations in the number, in particular of mononuclear alveolar macrophages, can be found.
3. No significant increase in the number of alveolar macrophages or polynuclear giant cells can be observed in cases of short or protracted suffocation. The duration of the asphyxiation process, at least in most of these forensic autopsy cases, does not seem to be long enough to enable the development of pulmonary polynuclear giant cells as described in experimental animals with considerably longer hypoxic agony.

## Immunoprofiles of alveolar macrophages and other markers in diagnosis

In 1964 Janssen and Bärtschi [23] described a swallowing and loosening of cytoplasm and nuclei, a perinuclear achromatosis and a degeneration of pseudopodia as well as an enlargement of alveolar macrophages after an interrupted oxygen supply of 30–90 minutes, taking these cellular changes as evidence of the asphyxia-triggered activation of this inflammatory cell line. Subsequently, the lack of any significant differences in the populations of alveolar macrophages in asphyxiated lungs vs lungs of control cases has called attention to the determination of the asphyxia-triggered recruitment state of residential alveolar macrophages. The availability of new research methods, especially immunohistochemistry, has shifted the focus onto the identification of cellular parameters, appearing specifically after asphyxia and growing in their expression level with the duration of suffocation.

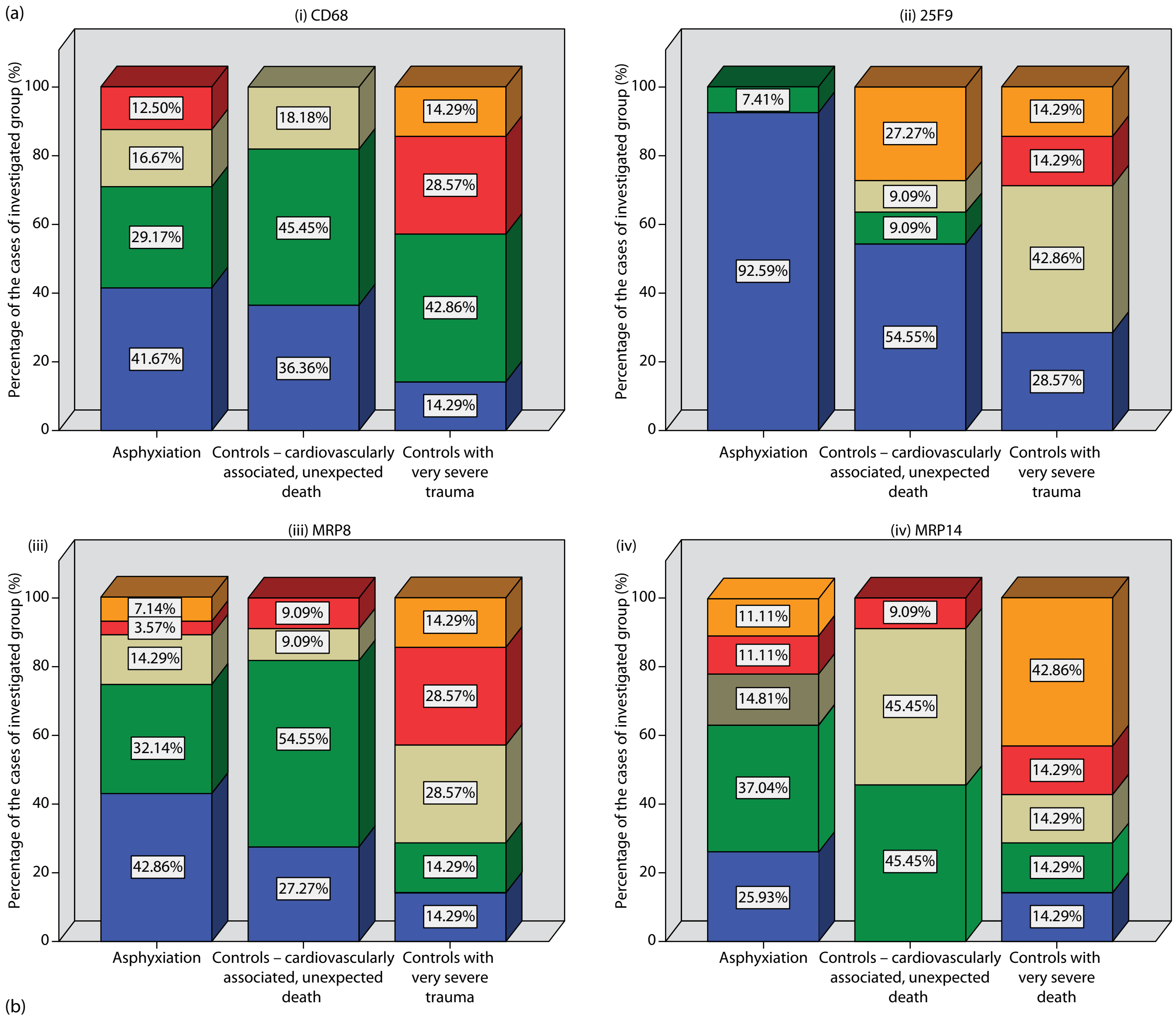
In their immunohistochemical characterization of the alveolar macrophages of the lungs of 22 opiate-involved deaths, 10 fatal strangulations and 10 cardiovascular controls, Grellner and Madea [18] assumed different antigenic profiles of macrophages, responding to asphyxia. They proved a strong immunohistochemical reactivity against the cytoplasmic macrophage marker LN-4 only in interstitial and perivascular macrophages, whereas all alveolar macrophages featured an expression of the general macrophage marker CD68 (PG-M1).

Investigating the question of different macrophage subpopulations and their significance in hypoxic inflammatory processes, Zwadlo et al. [41] reported on the so-called early-stage markers MRP8 and MRP14 and their heterodimer 27E10 as well as on the so-called late-stage marker 25F9 in 1985. MRP8 and MRP14 (migration inhibitory factor-related protein) are cytoplasmic calcium-binding proteins of the S100 protein family, responsible for the reorganization of the cytoskeleton of activated monocytes and playing a regulatory role in the modulation of the kinases, for example in the context of diapedesis and adhesion of leucocytes [32]. The heterodimer 27E10 is, in contrast to the intracellular localization of the monomers, expressed on the cell surface of subpopulations of macrophages, monocytes and granulocytes and reaches its maximal expression on the second and third day in stimulated monocyte cultures, diminishing thereafter [3]. The late-stage marker 25F9 is a typical epitope of mature macrophages, lacking in young monocytes as well as in all other blood cell lines. According to Zwadlo et al. [41], the expression of this epitope increases from the third day and reaches the maximum 8–9 days after the macrophage stimulation under cell culture conditions.

Recently, multiple studies have concentrated on observation of the expression level of these three markers in young macrophages, being activated by terminal asphyxia and dependent on the duration of the agony. In 1996, Du Chesne et al. [13] showed a doubling of MRP8 and MRP14 interstitial cell counts in protracted asphyxiation ($n = 8$) vs in control cases of hanging (an equivalence of a short asphyxiation ($n = 6$)) and peracute deaths ($n = 9$)). Observing a clear increase of 27E10-positive cells in the lung parenchyma after a protracted asphyxiation, they evaluated these three immunohistochemical markers as an additional diagnostic criterion for the differential diagnosis between acute and protracted asphyxiation. Focusing on a comparison of the myelomonocytic subtypes in the lungs of drowning victims vs lungs of hanging, sudden cardiac and immediate traumatic deaths as well as of fatal cerebral haemorrhages, Brinkmann et al. [5] also confirmed an asphyxia-triggered increase in the monocytic expression levels of MRP8 and MRP14, although higher levels were defined for cases of drowning.

In 2010, Strunk et al. [36] reported on the rising significance of the number of MRP8- and MRP14-positive early macrophages with the duration of asphyxiation. In their study of the human lung parenchyma of long protracted asphyxia ($n = 6$) with a suffocation of >25 minutes and of short protracted asphyxia with a suffocation of 10–25 minutes ($n = 8$) vs cases of immediate traumatic deaths ($n = 9$), they went on to estimate the existence of additional cofactors, accelerating the recruitment of young macrophages with the epitopes mentioned above. Whereas Zwadlo et al. [41] reported on the hypoxia duration of several days needed for the beginning of the expression of these epitopes, around 30 minutes of asphyxia seemed sufficient to reach even higher expression levels in Strunk's cases. As described in Chapter 16, the release of catecholamines, phospholipids [20,36] and the MCSF (macrophage colony stimulation factor) [21] as well as the expression of the urokinase plasminogen receptor [26], induced by hypoxia, have an additional stimulating effect on the maturation and activation of young monocytes in the organism, compared to cell culture conditions, lacking any supporting co-triggers. There is no doubt that several other co-triggering factors exist in a complex organism and are still to be investigated. Their overwhelming release under the condition of maximal stress explains an extremely high increase of MRP8/MRP14 levels in traumatic control deaths in our study (Figure 14.3a). Recently, the role of MRP8/MRP14 as a predictor of cardiovascular events due to atherosclerotic vascular injury was identified [12]. Disqualifying the case group of fatal cardiovascular deaths as a control, we could still demonstrate an upregulation of MRP8 and MRP14 in suffocation deaths with long asphyxia, showing a twofold (MRP14) and even a fourfold increase (MRP8) in comparison to short asphyxia [19].

In contrast to an obvious asphyxia-dependent upregulation of MRP8 and MRP14 in alveolar monocytes, recent data from literature concerning the expression levels of the late-stage marker 25F9 are controversial: for example, Grellner and Madea [17] showed a positive immunostaining against this epitope in 70 per cent of opiate-involved fatalities and strangulations and in 40 per cent of control cases. They used this observation as an argument for a longer pre-existence of alveolar macrophages and giant cells in the pulmonary tissue of asphyxiated persons and a reason to refute an agonal immigration, mobilization or proliferation of macrophages, proposed by Janssen et al. In our recent study [19], we reported on the detection of low levels of 25F9 in all case groups (Figure 14.3a). The differences in the results of these studies can be explained by a different study design: Whereas any chronic inflammation and accumulation of siderophages due to chronic congestion because of cardiovascular co-morbidities were excluded by a histologic pre-evaluation of the lung tissue and by a Prussian blue staining, Grellner and Madea did not make this pre-selection. Thus, we consider that the late-stage marker 25F9 to be inept as an asphyxiation marker as it



(b)

| Grading | | CD68 (cells/18 mm² × 200) | 25F9 (cells/18 mm² × 200) | MRP8 (cells/18 mm² × 200) | MRP14 (cells/18 mm² × 200) |
|---|---|---|---|---|---|
| 1 | 🟦 | 0–300 | 0–50 | 0–200 | 0–300 |
| 2 | 🟩 | 300–600 | 50–100 | 200–400 | 300–800 |
| 3 | 🟨 | 600–900 | 100–150 | 400–600 | 800–1300 |
| 4 | 🟥 | 900–1200 | 150–200 | 600–800 | 1300–1800 |
| 5 | 🟧 | >1200 | >200 | >800 | >1800 |

**Figure 14.3** Immunohistochemical analysis of the expression of specific markers. (a) Results of the immunohistochemical scoring in percentage/group. The data are generated by the summation of total cell numbers in subpleural and central areas. (b) Score of the immunohistochemical grading (From Gutjahr E, Madea B. *Forensic Sci Int* 2019;**297:**315–325).

requires a hypoxia duration of several hours up to several days until the beginning of the increased expression.

Suffocation, causing an interruption of the air flow and various processes of counter-regulation, is associated with strong forced breathing and an over-excitement of the autonomic nervous system by mechanical asphyxia [40], requiring an alveolar surface integrity. In human lungs, the pulmonary surfactant protein A (SP-A), produced by type II alveolar cells, serves as a film on the surface of the alveoli to reduce the tension and to facilitate their unfolding during breathing. Consequently, its secretion is increased under hypoxic conditions, causing a respiratory deficiency syndrome in case of its deficiency. The usefulness of SP-A as a practical diagnostic marker of fatal mechanical asphyxia in forensic autopsy cases was investigated by Zhu et al. [40]. In their study, they proved a significantly increased intensity of SP-A staining in the intra-alveolar space, accompanied by many massive aggregates in approximately 60 per cent of 27 asphyxia cases, findings not observed in the control group of 16 cases of poisoning and peracute deaths [40]. Cecchi et al. [8] confirmed the same tendency for the formation of massive intra-alveolar precipitates of SP-A in 32.4 per cent of cases of mechanical asphyxia (grade III) compared to 18.4 per cent of control cases (grade I).

According to the Euler–Liljestrand mechanism, acute exposure to hypoxia finally results in vasoconstriction of the pulmonary arteries of apical sections of the lung. Therefore, it is expected that the acute inflammatory response to asphyxia of the lung involves mediators of the vessel response to an inflammatory insult and thus some of them may represent a good marker for forensic purposes [8]. P-selectin is a glycoprotein stored for rapid release in Weibel-Palade bodies of the endothelium and in alpha granules of platelets [24] and it represents an adhesion molecule involved in the process of leucocyte rolling on the endothelium. Having a similar adhesion function, E-selectin is expressed by endothelium only after a cytokine activation. In the skin, its expression is detected after 1–2 h after the insult [8]. Ortmann and Brinkmann [31] observed an overall occurrence of P-selectin in lung vessels with an intense homogeneous staining pattern in acute hypoxic, non-inflammatory deaths (hanging, carbon monoxide, cyanide intoxication). In their study, a slightly weaker intensity was shown for the cases of drowning, whereas protracted inflammation-associated deaths (pneumonia, septic shock) were characterized by an irregular distribution and a weak intensity of the staining. Similar results were described by Cecchi et al. [8]. Additional immunodetection in healthy lungs ($n = 11$) in this study, however, emphasized a diffused P-selectin expression with a mild and moderate intensity as an equivalence of a basal and constitutive expression of this marker of the lungs. This result prevents P-selectin from being used in the diagnosis of mechanical asphyxia. Because the same study presented comparable results for E-selectin expression, reaching similarly high levels in the tissue of healthy and asphyxiated lungs, E-selectin has also lost its validity as a potential asphyxia marker.

One of the most promising potential markers seems to be HIF-1α (hypoxia inducible factor-1α), a transcription factor expressed in response to hypoxia and later activating the expression of the genes involved in erythropoiesis, angiogenesis, modulation of vascular tone, etc. The active form of this transcription factor is the heterodimer HIF-1, consisting of the constitutively expressed HIF-1β-subunit and the HIF-1α-subunit, which is constitutively produced, but degraded under normoxic and accumulated under hypoxic conditions in mammalian cells in a time- and $O_2$ concentration-dependent matter [34]. In their rat experiments, Yu et al. [39] showed a maximally high expression of HIF-1α in the bronchial epithelium, bronchial smooth muscle, alveolar epithelium and vascular endothelium after ventilation with 0–1 per cent $O_2$ for 4 hours. This effect could be reversed after reoxygenation, causing a degradation of HIF-1α. Following these findings, Cecchi et al. [8] proved a similar tendency in the blood vessels of asphyxiated lungs. In their study of 34 cases of acute mechanical asphyxia and 28 control cases, including hanging, they detected an expression of HIF-1α in all vessel types of mechanically asphyxiated lungs but no significant expression of HIF-1α in normal conditions. Moreover, the cases of hanging, often associated with hypoxaemia, even showed higher expression levels of this epitope.

In conclusion, the evaluation of the immunoprofile of alveolar macrophages and the determination of certain immunomarkers of lung vessels can be useful in diagnosing the asphyxia-associated mechanism of death in individual cases. The following aspects should be considered in the forensic routine:

1. In individual cases, the determination of the recruitment state of alveolar macrophages can be helpful, although not absolutely specific, in diagnosing suffocation. Normally, an asphyxia duration of approximately 20–30 minutes is sufficient to induce changes at the molecular level. The late-stage inflammatory marker 25F9 can be used to discriminate the accumulation of pre-existing mature macrophages in the context of chronic diseases (e.g. COPD, chronic cardiac insufficiency). The expression level of the cytoplasmatic early-stage inflammatory markers MRP8 and MRP14, as well as of their cell-surface heterodimer 27E10, increases with the duration of pre-terminal asphyxia and can be used to distinguish between a short/acute and long/protracted asphyxiation.
2. Intra-alveolar granular deposits of SP-A seem to be related to an intense hypoxic stimulus and can suggest, together with other elements, a severe hypoxia as the mechanism of death (in case of massive deposits).
3. P- and E-selectin expression in the lung vessels, activated by several types of trigger stimuli, cannot be used as an indicator of asphyxia.
4. HIF-1α seems to have a high potential for forensic utility. It is expressed in small-, medium- and large-calibre lung vessels of the vast majority of mechanical asphyxia deaths and shows increasing expression levels with a rising duration of pre-terminal hypoxia. As its half-life time is relatively short, however, this marker can only be considered in cases with a short post-mortal period because of its rapid degradation.
5. Despite a hypoxia-associated expression, all the immunohistochemical markers that have been studied so far are non-specific and thus require cautious use as forensic proof of suffocation as a cause

of death. In addition, the high costs and extensive time needed for the performance of analysis prevent a broad application in forensic routine.

## Mast cell activation and mast cell tryptase in diagnosis

Whereas alveolar macrophages were the main protagonists of forensic science in the context of protracted asphyxia in the last decades, mast cells attracted little research interest, primarily because of their obvious role in anaphylaxis and drug-associated deaths. However, several studies of the chronically hypoxic lungs (e.g. in case of asthma, COPD, pulmonary hypertension) emphasize the function of mast cells in hypoxia-triggered tissue remodelling, angiogenesis as well as tumour growth. Tucker et al. [37] described significant changes in the density of mast cells in the lungs of chronically hypoxic pigs, rats and sheep. In their investigations of the lung specimens of 35 asphyxia/hypoxia human deaths and 11 controls, Muciaccia et al. [28] confirmed that short periods of lack of oxygen (a few minutes only) resulted in large quantities of mast cells, rapidly recruited in the lungs and accumulating in perivascular areas. In contrast, using a toluidine-blue staining in our study of 13 short-asphyxiated and 15 long-asphyxiated lungs vs the lungs of 11 sudden cardiovascular fatalities and of 7 traumatic deaths, we observed approximately equal levels of mast cell numbers in the cases of suffocation with prolonged asphyxia and of both control groups (Figure 14.2b). Only the lung parenchyma of suffocation deaths with a short agony was characterized by a slightly lower mast cell population [19]. Our conclusion is that the forensically observed duration of asphyxiation is too short to induce a significant increase in activated pulmonary mast cells. In fact, Muciaccia et al. [28] reported elevated mast cell numbers in cases of contact of the alveolar and bronchial walls with foreign antigens in deaths due to aspiration and anaphylaxis only, suggesting an additional stimulus besides hypoxic conditions.

Although an increase in mast cell numbers does not seem probable during a normally observed suffocation scenario, Nadziejko et al. [30] demonstrated an effect of the alveolar hypoxia on pre-existing mast cell populations. After exposure to a localized alveolar hypoxia, they observed a decrease of 12 per cent in the granule content of perivascular mast cells. These results concentrate the attention on the recruitment state of pre-existing mast cells rather than on the hypoxia-triggered immigration of the mast cells to the lung. These tendencies correspond to the observations concerning the hypoxia-triggered activation of pre-existing alveolar macrophages and their molecularly measurable recruitment changes, as described above.

When taking a closer look at the pre-existing alveolar inflammatory cell populations, the interactions of alveolar macrophages and pulmonary mast cells should be highlighted. Interestingly, Chao et al. [9] not only provided direct evidence of an acute hypoxia-induced activation of macrophages, but also analysed the interaction of alveolar macrophages and mast cells in their studies in primary cell cultures of these two cell lines. In these studies, the exposition of alveolar macrophages to $pO_2$ values of <30–35 Torr (normoxia ≈100 Torr) resulted in a respiratory burst, confirmed by a release of $H_2O_2$ into the supernatant within 15 minutes of hypoxia. The addition of the supernatant from the alveolar macrophage culture to a primary culture of mast cells resulted in their degranulation. When no degranulation of mast cells in primary cell culture was triggered by simple acute hypoxia in the same studies, monocyte chemoattractant protein-1 (MCP-1), a mast cell secretagogue released by alveolar macrophages, was identified, indicating that alveolar macrophages are affected by acute hypoxia [9]. Therefore, MCP-1 secretion from alveolar macrophages induces mast cell activation, which leads to microvascular inflammation via increased leucocyte–endothelial adhesive interactions, leucocyte emigration and vascular permeability by the activation of renin–angiotensin system (RAS) [10].

Among the substances being released by the degranulation of mast cells, mast cell tryptase, a serine protease stored in the mast cell granules and released together with histamine, is relatively stable (long half-life) and can be found in blood from a few minutes up to several hours after secretion [33]. Edston et al. [14] found an increase of the mast cell tryptase concentrations in the femoral blood of the cases of acute mechanical asphyxia. Fineschi et al. [16] documented a similar increase in heroin-related deaths, being accompanied by a terminal acute hypoxia. Using mast cell tryptase as a representative parameter to show the degranulation of mast cells due to hypoxia, we measured higher levels of tryptase in suffocation deaths compared to sudden cardiovascular fatalities in our recent study [19] (Figure 14.4). This result

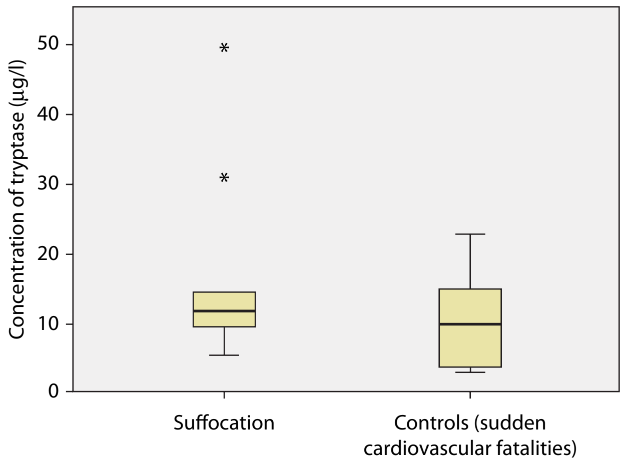


**Figure 14.4** Concentration of mast cell tryptase in femoral blood of asphyxia-associated deaths and controls (sudden cardiovascular fatalities) (From Gutjahr E, Madea B. *Forensic Sci Int* 2019;**297:**315–325).

proves an achievement of the early stage of hypoxia-induced inflammation, probably followed by the recruitment of mast cells in case of longer survival periods than observed in our study. So far, no standards for the measurement of the mast cell tryptase in postmortal femoral blood, including confounders and standard values in healthy lungs, have been established. Hence, we cannot make any conclusions on the severity of the elevation of tryptase concentrations in asphyxia-associated deaths. Despite the lack of statistical significance and a slight difference from the mean tryptase concentration in cardiovascular control cases only, we exclude the randomness of the asphyxia-associated tryptase elevation in consideration of the high concentrations of serum tryptase in cases of acute cardiovascular disease, reported by Filiapak et al. [15]. Even regarding tryptase levels in cardiovascular fatalities as non-elevated implicates a performed mast cell degranulation in suffocation deaths. Given the small number of cases in our study (nine cases of fatal suffocation and nine control cases of cardiovascular fatalities) as well as potential confounders, which we partly tried to exclude (e.g. coronary atherosclerosis), no general conclusions can be drawn concerning the diagnostic value of mast cell tryptase so far. Further investigations with larger collectives and an identification of additional confounders should follow to verify mast cell tryptase levels in postmortal femoral blood as a significant marker of hypoxia-triggered processes.

Research should undoubtedly continue to focus also on the identification of the MCP-1 levels in femoral blood depending on the asphyxia period. Functioning as a mediator of mast cell activation by alveolar macrophages, MCP-1 concentration in blood could potentially confirm the propagation of the inflammatory cascade from alveolar macrophages to mast cells, even before an increase of mast cell numbers can be observed. However, the rapid postmortem proteolysis of MCP-1 unfortunately poses a challenge in these investigations.

In summary, a closer look at mast cell numbers and their recruitment state can help to identify an extremely long duration of preterminal asphyxiation. However, the following aspects have to be considered:

1. Even the prolonged duration of preterminal asphyxia normally observed in forensic cases is insufficient to induce an immigration of mast cells into the lung parenchyma.
2. However, a prolonged asphyxia induces the release of MCP-1, a mast cell secretatogue, by alveolar macrophages. The measurement of the concentrations of MCP-1 in the blood could potentially allow conclusions to be drawn concerning the asphyxia duration, even before an activation of mast cells as the next link of the inflammation chain. Unfortunately, the short half-life of MCP-1 hinders further investigations and the use of MCP-1 concentration in forensic routine.
3. An increasing duration of the preterminal asphyxia is coupled with an activation of mast cells and their degranulation. Measuring mast cell tryptase levels in postmortem samples of femoral blood can be used as a parameter for this degranulation. So far, no standards for these measurements (i.e. confounders, normal concentration in the blood of healthy persons, technical conditions) have been established.

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# 15 Molecular Pathology

*Toshikazu Kondo*

## Introduction

Oxygen deficiency due to asphyxiation can cause hypoxic damages in every organ. Many forensic pathologists have studied hypoxia-specific damages in several organs [15]. The susceptibility of organs and tissues to oxygen deficiency varies, depending on the degree of differentiation, oxygen requirement and functional condition of the cells. It is apparent that oxygen deficiency results in the induction of various molecules at gene and protein levels in the parenchymal and mesenchymal cells of each organ in order to adapt or overcome such hypoxic stress.

Conventional histopathological application is the most common forensic practice for the diagnosis of asphyxiation, but it is sometimes insufficient. In order to improve accuracy and objectivity, advanced methods are applied to forensic samples with postmortem changes [9,17]. As shown in Tables 15.1 and 15.2, biochemical, immunohistochemical and molecular biological techniques are employed for diagnosis of mechanical asphyxiation.

## Brain

### Oxygen-regulated protein 150 (ORP150)

ORP150, a member of the heat-shock protein (HSP) family, plays a crucial role as a molecular chaperone to prevent irreversible changes in immature proteins as a result of various types of stimulation. In particular, hypoxic conditions have been found to induce ORP150 expression, which is localized in the endoplasmic reticulum (ER) of the cerebral neurones, with the intensity of ORP150 expression dependent on the duration of brain ischaemia. These observations imply that immunohistochemical and morphometric analysis of ORP150 in the brain may be one of the clues to diagnosing mechanical asphyxiation in forensic autopsy cases [10].

### HSP70

Kitamura [16] focused on the hypoxia-induced alterations in CA1 pyramidal cells of the hippocampus and neurones in the 3rd to 6th neocortical layers, which are susceptible to oxygen deficiency. Protein HSP70 expression was found in the CA2, CA3 and CA4 regions in cases of long-term survival after severe hypoxic/ischaemic injury. Thus, it is suggested that the detection of HSP70 in the hippocampus indicates hypoxic/ischaemic injury or other stress prior to death. In forensic practice, immunohistochemical investigation of HSP70 can be of great value for diagnosing not only hypoxic/ischaemic brain damage during the process of death but also the victim's past history of hypoxic attacks.

### Ubiquitin (Ub)

Ubiquitin is a highly conserved protein in all eukaryotes, and it seems to be involved in neurodegenerative diseases and damage induced by chronic ischaemia. Immunohistochemically, ubiquitin was localized at the nuclei of pigmented substantia nigra neurones, with two staining patterns – inclusion and diffuse types. In particular, both staining patterns were more evident in mechanical asphyxiation (strangulation and aspiration/choking) except for hanging and drowning strangulation, aspiration/choking and drowning. Collectively, intranuclear ubiquitin immunoreactivity of the pigmented substantia nigra neurones in the midbrain appear to be a hallmark in asphyxiation and drowning [22].

### Astrocytes and related proteins

Astrocytes are more resistant to hypoxic conditions than neurones. The S100 proteins have an A and B subunit and are small acidic calcium-binding proteins. S100B is highly specific for astrocytes, oligodendrocytes and ependymocytes in the central nervous system. Clinically, S100B seems to act as a serum marker of brain damage from cerebral injury and hypoxia/ischaemia. Li et al. [20] found that, in cases of asphyxiation due to neck compression, the number of astrocytes immunostained with anti-S100 or anti-GFAP was significantly decreased, compared with that for other asphyxiation and acute myocardial infarction. Reciprocally, serum S100B levels were significantly higher in asphyxiation by neck compression than in other types of asphyxiation. These observations imply that astrocytes and serum S100B would be available biomarkers for supporting the diagnosis of asphyxiation due to neck compression.

## Lungs

### Alveolar macrophages

Janssen reported several specific hypoxia-induced findings in the lungs [15]. Briefly, there was abnormal migration of

**Table 15.1 Molecular and cellular markers for mechanical asphyxiation in the organs**

| Markers | Organs | Application |
|---|---|---|
| ORP-150 | Brain (cortex) | IHC |
| HSP70 | Brain (hippocampus) | IHC |
| Ub | Brain (midbrain) | IHC |
| Astrocytes | Brain | IHC |
| Macrophage | Lung | HE , IHC |
| Mast cells | Lung | IHC |
| P- and E-selectin | Lung | IHC |
| HIF-1 | Lung | IHC |
| Surfactant | Lung | IHC, GE |
| ANP | Heart | GE |
| BNP | Heart | GE |

IHC: Immunohistochemistry; HE: Haematoxylin and Eosin staining; GE: mRNA analyses.

**Table 15.2 Molecular markers for the determination of vitality in compressed neck skin**

| Markers | Localization |
|---|---|
| CD15 | Dermal connective tissue |
| IL-15 | Around the dermal vessels |
| Tryptase | Dermal connective tissue |
| Cathepsin D | Intradermal cells |
| P-selectin | Intradermal cells and vessels |
| AQP3 | Epidermal cells |
| Enkephalin | Carotid body |
| VIP | Carotid body |

macrophages in the alveoli of the lungs in young persons who had died from mechanical asphyxiation. Moreover, they demonstrated that the migrated macrophages were transited into polynuclear giant cells in experimental animals dying of prolonged hypoxia with a duration of between 30 minutes and 12 hours [15], thus indicating that the detection of those polynuclear cells in the lungs would be available for the histopathological diagnosis of asphyxiation due to strangulation (Figure 15.1). In addition, macrophages are a heterogeneous lineage, and classified into early and late stages of inflammation. The detection of macrophage subtypes might therefore be useful for differential diagnosis of acute and protracted asphyxiation [5].

Betz et al. [1] carried out immunohistochemical investigations on alveolar macrophages (with one or two nuclei) and polynuclear giant cells (defined as alveolar macrophages containing three or more nuclei). Immunopositive reactions for Ki-67, one of the proliferation marker antigens, could not be observed in polynuclear giant cells, suggesting that those cells had not quickly developed before death by endomitosis as an adaptive reaction following oxygen deficiency. There was no significant difference in the number of alveolar macrophages or polynuclear giant cells between asphyxiation and control groups. Thus, the detection of alveolar macrophages and/or polynuclear giant cells is not useful for diagnosing asphyxiation.

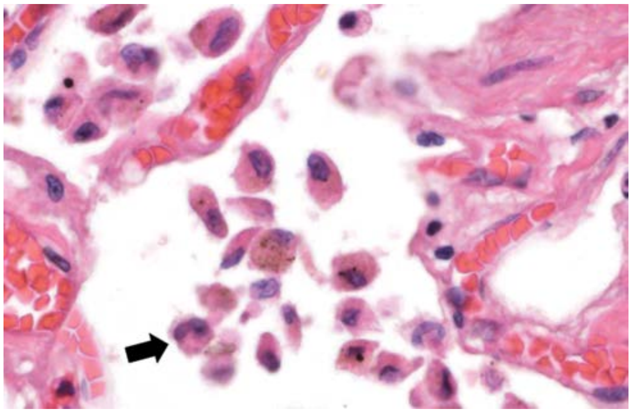

**Figure 15.1** Intra-alveolar mononuclear macrophages and polynuclear macrophages (arrow) in strangulation death (38-year-old male) (HE staining, ×400).

## Mast cells

From the point of view of forensic pathology, mast cells and their related molecules seemed key molecules for diagnosis of anaphylaxis [6] or wound vitality [2,7]. Moreover, Edston and colleagues demonstrated that mast cell-derived tryptase levels in the blood were elevated in asphyxiation deaths [7]. CD117 (c-kit) is one of the specific markers for mast cells, and Cecchi and colleagues [3] examined the immunohistochemical dynamics of intrapulmonary mast cells through the detection of c-kit. C-kit+ mast cells was significantly in evidence in the lungs of asphyxia deaths, such as hanging, strangulation and aspiration deaths (Figure 15.2). These observations imply that the detection of intrapulmonary mast cells would be a good tool for the differentiation between acute asphyxia deaths and other kinds of death [21].

## Selectins and HIF

It is well known that several biological pathways are initiated after hypoxic pulmonary damage. A comprehensive study

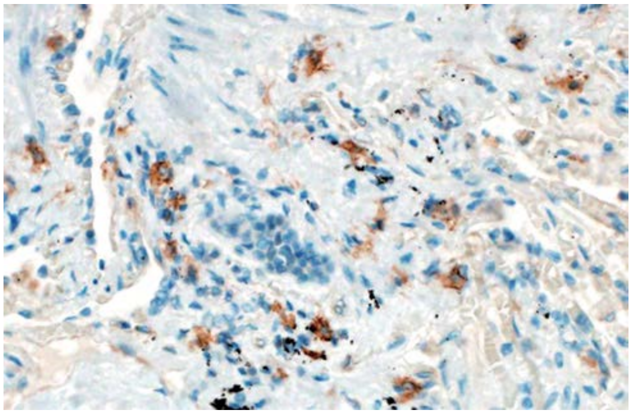

**Figure 15.2** Immunohistochemical detection of c-kit+ mast cells in the perivascular region of the lungs (45-year-old male, hanging) (×200).

has been carried out on vital reactions of lung tissue to asphyxiation with special regard to P- and E-selectins, SP-A and HIF1-α. The expression of P- and E-selectins was enhanced in intrapulmonary vessels after several types of trigger stimulus, including hypoxia, thus implying that these molecules could not be applied as a specific indicator of asphyxiation. HIF1-α is a transcriptional factor produced in response to hypoxic conditions that activates gene expression involved in erythropoiesis, angiogenesis, glycolysis and modulation of vascular tone. HIF1 is a heterodimer composed of HIF1-α and HIF1-β subunits. HIF1-α accumulates in the nuclei of mammalian cells under hypoxic conditions in an $O_2$ concentration-dependent manner. Experimental studies using rats suggested that the gene expression of HIF1-α but not HIF1-β was upregulated in pulmonary artery endothelial cells under hypoxic conditions. Cecchi and colleagues [3] examined HIF1-α expression immunohistochemically in the lungs of asphyxiation death. They found that there was increased expression of HIF1-α in the intrapulmonary vessels in asphyxiation cases, suggesting an interesting potential use of HIF1-α as a screening test for asphyxia deaths [3].

### Pulmonary surfactant

Pulmonary surfactant is a surface-active lipoprotein (phospholipoprotein) produced by type II alveolar cells and composed of 80 per cent dipalmitoylphosphatidylcholine and surfactant-associated proteins (SP-A, B, C and D). SP-A is the major surfactant protein and its deficiency is known to cause infantile respiratory distress syndrome. Zhu and colleagues [26] found specific immunostaining patterns such as massive aggregates of SP-A-stained granules and intense and diffuse SP-A-positive staining with many massive aggregates in mechanical asphyxiation deaths.

SP-A is coded by two different genes, *SP-A1* and *SP-A2*, which have highly similar sequences. Ishida and colleagues [13] examined the gene expression of SP-A1 and SP-A2 to the lungs obtained from forensic autopsy cases. The SP-A1 : SP-A2 ratio was significantly elevated in mechanical asphyxiation and drowning compared to control groups. However, there was no significant difference between mechanical asphyxiation and drowning. These observations imply that analysis of the SP-A1 : SP-A2 ratio would lend support to diagnosing mechanical asphyxiation.

## Heart

Atrial and brain natriuretic peptides (ANP and BNP) are clinically utilized for the evaluation of cardiac function. From the forensic perspective, the intracardiac gene expression of ANP and BNP has been analysed in autopsy cases. The expression of ANP and BNP mRNA was found to be lower in mechanical asphyxiation and drowning than in sudden cardiac deaths. These observations suggest a difference between primary heart failure in sudden cardiac death and terminal cardiac dysfunction secondary to fatal asphyxiation or drowning [4].

## Wound vitality of neck compression

Forensic pathologists are frequently required to determine whether compression marks on the neck are associated with wound vitalities. Metachromasia in dermal collagen fibrils can be observed in both antemortem and postmortem neck compression marks, which makes it difficult for the determination of vitality in compressed neck skin specimens (Figure 15.3).

### Cytokines

In damaged skin lesions, leucocytes such as neutrophils and macrophages are recruited under the guidance of cytokines and chemokines, implying that those cytokines and chemokines could be available markers for wound vitality. Turillazzi and colleagues [25] immunohistochemically examined the expressions of several cytokines and inflammatory mediators in neck skin specimens obtained from autopsy cases of death due to hanging, to discuss their significance in assessing whether hanging marks and signs occurred before or after the death of the victim. Their investigations demonstrated that tryptase, IL-15 and CD15 would be reliable for the determination of vitality in ligature marks on the neck.

### Metallic ions

A unique study carried out by Legaz Pérez et al. [19] analysed the concentrations of metallic ions (Fe, Zn, Mg and Ca) and the expression of P-selectin and cathepsin D in skin ligature marks. They found that intradermal

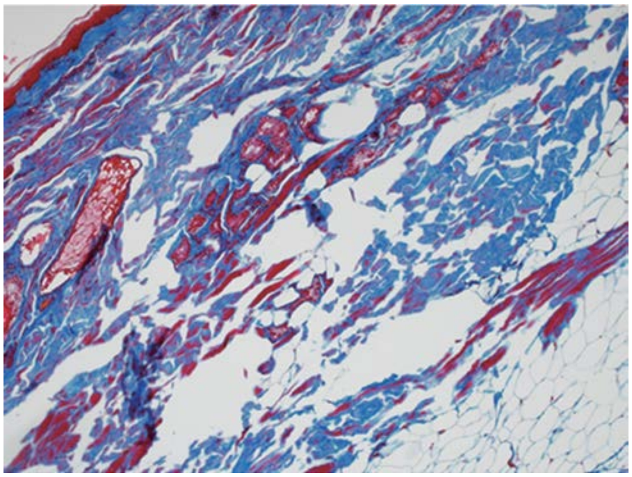

**Figure 15.3** Masson's trichrome staining demonstrates metachromasia of the dermis in the neck skin of strangulation death (65-year-old female) (×100).

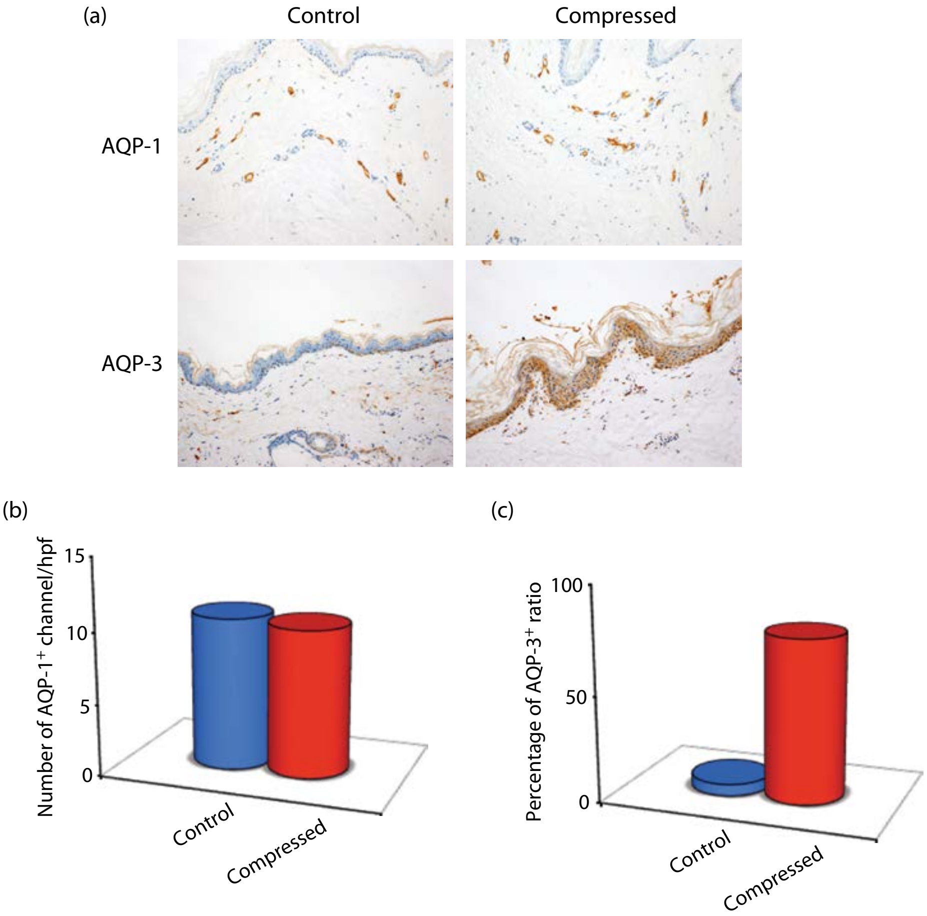

**Figure 15.4** The expression of AQP1 and AQP3 in the skin. (a) Immunohistochemical detection of AQP1 and AQP3 in uninjured skin and compressed neck skin (×400). (b),(c) Semi-quantitative analyses of AQP1 and AQP3 (Cited from Ref [14] with kind permission of Springer).

concentrations of Ca and Mg were higher than those of Fe and Zn, although there was no significant difference in the concentrations of Ca and Mg between uninjured skins and ligature marks. The Fe and Zn concentrations, however, were significantly elevated in ligature marks. Moreover, there were positive correlations between cathepsin D and Fe and between P-selectin and Fe. Low Fe levels were observed in the case of a negative expression for cathepsin D and P-selectin, whereas high levels of Fe were found in sections with strong immunoreactivity cathepsin D and P-selectin. No significant relationships were found between Ca, Mg and Zn and the expression of cathepsin D or P-selectin.

## Aquaporins

Aquaporins, a family of channel proteins, play important roles in water and fluid transport in regulation of osmolality in the body. Although aquaporin 1 (AQP1) was immunostained in dermal capillaries in both neck compression marks and intact skin samples, a recent study found no significant difference in AQP1 expression between the two groups (Figure 15.4) [14]. On the contrary, aquaporin 3 (AQP3)-positive signals seemed more intense in the keratinocytes in compression regions (Figure 15.4). From a forensic pathology viewpoint, immunohistochemical detection of AQP3 in neck skin can be considered a valuable marker to diagnose antemortem compression.

## Carotid body

The carotid body is a small cluster of chemoreceptors and supporting cells located near the fork (bifurcation) of the carotid artery (which runs along both sides of the throat). It is easily recognized that the carotid body is mechanically and chemically stimulated when the neck is compressed. However, for many years little work had been done on the carotid body for diagnosing asphyxiation. In 1994 Kubo and colleagues [18] reported on an immunohistochemical investigation of carotid bodies in forensic autopsy cases. In their study, chief cells were more lightly stained with anti-neuropeptides (enkephalin and VIP) and their size was enlarged in neck compression cases. These observations imply that immunohistochemical investigation of the carotid body would be useful for diagnosing neck compression.

## Postmortem biochemistry in asphyxiation

In clinical practice, biochemical analyses using blood samples are routinely performed. Recently, those analyses have been employed in forensic autopsy cases in order to support the diagnosis of the cause of death. However, the majority of biochemical markers are difficult to apply to forensic autopsy cases because of postmortem changes. In diagnosis of asphyxiation by neck compression, thyroid hormone is raised as one of the biochemical markers. In particular, it has been suggested that the serum levels of thyroid hormone (T3 and thyroglobulin) are elevated in asphyxiation, indicating that they might be indicators of vital reaction in neck compression [23]. Subsequently, it has been recommended that peripheral arterial blood situated distant from the thyroid gland be used for the measurement of thyroglobulin [8]. However, my own study with colleagues has demonstrated that the elevation of serum thyroglobulin is not specific for asphyxiation by neck compression, indicating that serum thyroglobulin is not reliable for diagnosing the death of asphyxiation by neck compression (unpublished data).

### Comprehensive analysis of gene expression in mechanical asphyxiation

In order to identify new biomarkers for the diagnosis of mechanical asphyxiation and neck compression, comprehensive analysis of gene expression has been performed in the brain and lung and neck skin. In forensic practice, biological reactions, known as ‘supravital reactions’, can proceed in the body from somatic death to cell death. Immediate early genes (IEGs) such as c-fos, fos-B and c-jun have been found to be significantly upregulated in the brain and lungs of mice sacrificed by mechanical asphyxiation [9]. Subsequently, several novel hypoxia-specific genes have been identified in the brain and lungs of mice after extended hypoxic conditions with suffocation, thus indicating possible gene markers for the diagnosis of mechanical asphyxiation [12,24]. Moreover, for the determination of vitality in compressed neck skin, several candidate genes have been detected [11]. The results may contribute to clarifying the pathophysiology of compression of the skin and may be useful in the diagnosis of asphyxiation.

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# 16 Biochemistry

*Cristian Palmiere*

## Introduction

Postmortem biochemical investigations in forensic cases of external neck compression have been proposed by some research teams to identify laboratory evidence supporting the hypothesis of antemortem mechanical asphyxia in association with morphological findings obtained from autopsy and histology. Most of the studies performed focused on hormone and hormone precursor leakage from thyroid and parathyroid glands potentially caused by application of mechanical force in the neck area.

A general description of thyroid and parathyroid hormone synthesis and secretion will be provided in this chapter along with some literature reviews. The clinical literature review focused on changes in blood thyroid hormone concentrations following generalized and localized trauma to the neck region while the forensic literature review targeted postmortem behaviour of thyroglobulin, thyroid hormones, thyroid-stimulating hormone and parathyroid hormone. An exhaustive review of forensic literature covering postmortem biochemistry application in the diagnosis of mechanical compression of the neck region inflicted prior to death is also considered in this chapter.

## Thyroid hormone synthesis

The thyroid gland, bilobular in structure and located at the front of the neck, is made up of two types of endocrine cells called follicular and parafollicular cells. The former (also called thyrocytes) are responsible for thyroglobulin (Tg) synthesis. The latter (also known as C-cells) secrete the hormone calcitonin, which is involved in calcium homeostasis [12].

Tg, a homodimeric glycoprotein made up of two 330 kDa chains, plays a fundamental role in the synthesis of thyroid hormones 3,5,3′-triiodo-L-thyronine (T3) and 3,5,3′,5′-tetraiodo-L-thyronine (thyroxine or T4). After synthesis, Tg is transported and stored in the follicular lumen. The thyroid follicles comprise a monolayer of polarized thyrocytes with the baso-lateral surface facing the bloodstream and the apical surface delimiting a central, spherical follicle lumen. The lumen is filled with 'colloid' (mainly highly concentrated Tg in different stages of oligomerization). Iodine enters thyroid follicular cells as inorganic iodide and is transformed into its organic form by the iodination of specific tyrosyl residues of Tg to form monoiodotyrosine (3-iodotyrosine, MIT) and 3,5′-diiodotyrosine (DIT). Thyroid peroxidase is the key enzyme in thyroid hormone formation as it catalyzes both the iodination of specific tyrosyl residues of Tg and coupling MIT and DIT to produce T3 and T4. Highly iodinated Tg is removed from the follicular lumen by micropinocytosis and undergoes proteolytic cleavage to release T3 and T4, which are then secreted into the bloodstream [23,25,26,46,58].

The thyroid gland produces two main iodothyronines, T4 (the pro-hormone) and T3 (the biologically active thyroid hormone). In humans, T4 is synthesized entirely within the thyroid gland and acts as a pro-hormone to generate T3. Circulating T4 is derived from thyroid gland secretion, whereas most (80%) of the pool of systemic T3 is generated by T4 monodeiodination in peripheral tissues. Only 20 per cent of T3 in the bloodstream is secreted directly by the gland itself [61].

T3 and T4 synthesis and release are controlled and regulated by a classical 'negative feedback system'. This involves reciprocal interactions between the thyroid gland and higher levels of control using information conveyed by circulating hormones. In this system, referred to as the hypothalamic–pituitary–thyroid axis, thyrotropin-releasing hormone (TRH) is secreted by the hypothalamic paraventricular nucleus into portal circulation, acting on the anterior pituitary thyrotropes to stimulate thyrothropin (thyroid-stimulating hormone, TSH) release. This subsequently acts via TSH receptors (TSHR) on thyroid follicular cells to stimulate cell proliferation as well as T4 and T3 synthesis and secretion [6,22,60,76].

Most circulating T4 and T3 in the blood is bound to specific carrier proteins knows as thyroid hormone distributor proteins. These include thyroxine-binding globulin (TBG), transthyretin (TTR, previously known as thyroxine-binding pre-albumin, responsible for much of immediate thyroid hormone delivery to tissues) and albumin, all of which are synthesized by the liver. In addition, a small fraction of thyroid hormones is distributed by lipoproteins. The concentrations of each of the thyroid hormone distributor proteins in adult human blood vary greatly: TBG at 0.015 g/l, TTR at 0.25 g/l and albumin at 42 g/l. Only approximately 0.2 per cent of total available T3 and 0.02 per cent of

T4 circulate as free unbound hormones (fT3 and fT4). The 'free hormone hypothesis' states that it is the free hormone in blood (not the protein-bound hormone) that is important for biological activity. Thus, thyroid hormones must dissociate from their distributor proteins before they can exert their effects. Circulating fT3 and fT4 ultimately act in the hypothalamus and anterior pituitary to inhibit TRH and TSH synthesis and secretion. This means that normal systemic, euthyroid status is maintained within a normal reference range by a negative feedback loop that establishes a physiological, inverse relationship between TRH, TSH and circulating fT3 and fT4, thereby defining the hypothalamic–pituitary–thyroid axis set point. Systemic thyroid hormone and TSH concentrations vary significantly among individuals, indicating that each person has a unique set point [6,22,57,60,76].

In extrathyroidal tissues, conversion of the pro-hormone T4 to the active form T3 (by iodine atom removal from the outer ring of T4), as well as T3 and T4 metabolism to inactive products (by removal of an inner-ring iodine atom) are mediated by three selenoenzymes called type 1, type 2 and type 3 deiodinase. All deiodinases are membrane-anchored proteins of 29–33 kDa that share substantial sequence homology and catalytic properties, and contain selenocysteine as the key residue within their catalytic centre. Expression levels and activities of type 1 deiodinase (D1 or DIO1, which is preferentially expressed in the thyroid gland, liver and kidney) and type 2 deiodinase (D2 or DIO2) vary among tissues, leading to tissue-specific differences in circulating levels of T3. Type 1 deiodinase catalyzes the removal of inner- or outer-ring iodine atoms in equimolar proportion to generate T3, 3,3′,5′-triiodothyronine (reverse triiodothyronine or rT3) or 3,3′-diiodothyronine (T2), depending on the substrate. Most of the circulating T3 is derived from T4 to T3 conversion by D1 action. Type 2 deiodinase, which is considerably more efficient than D1, catalyzes only outer-ring iodine atom removal from T4, generating the active product T3. D2 is mainly active in the brain, pituitary and skeletal muscle. Type 3 deiodinase (D3 or DIO3) irreversibly inactivates T3, or prevents T4 activation by catalyzing the removal of an inner-ring iodine atom to generate T2 or rT3 respectively. Given these functions, D3 is considered the major physiological inactivator and terminator of thyroid hormone action at the peripheral level [4,6,11,15,47,61,72,76].

It has been reported that the activities of D2 and D3 are designed to maintain local tissue T3 content as normal as possible in the face of altered serum hormone levels. In states of iodine deficiency and hypothyroidism, D2 activity is markedly upregulated and D3 activity is decreased to increase the proportion of T3 formed locally. This also lessens its degradation in order to maintain the available amount of T3 within the normal range. Opposite changes in D2 and D3 activity occur in hyperthyroidism [71].

## Calcitonin synthesis and functions

Thyroid parafollicular cells are the major producers of circulating calcitonin, a polypeptide hormone consisting of 32 amino acids, with a carboxyterminal proline amide and an N-terminal disulfide bridge connecting the cysteines at positions 1 and 7 to form a 7 amino-acid ring structure at the amino terminus. Calcitonin is initially synthesized as a precursor containing 136 amino acids and a leader sequence at its amino terminal region, which is cleaved during hormone transport to the endoplasmic reticulum. The basic function of calcitonin is plasma calcium regulation by a feedback mechanism. The effect in this regard is obtained through the inhibition of bone resorption, thus reducing the amount of circulating calcium. Calcitonin decreases blood calcium levels by direct inhibition of mediated bone resorption and enhancing calcium excretion by the kidney [5,33,37,38,48,54].

## Thyroid function tests

As stated above, T4 is entirely synthesized within the thyroid gland in humans and circulating T4 is derived from thyroid gland secretion. Total T4 serum concentrations in adults are in the range 5–12 μg/dl (64–154 nmol/l).

Serum total T3 concentration reflects the functional state of peripheral tissue rather than secretory performance of the thyroid gland. Total T3 serum concentrations in adults the range 90–190 ng/ml.

Normal serum fT4 values in adults range from 1.0 ng/dl to 3.0 ng/dl (13–39 pmol/l). A minute amount of thyroid hormone circulates in the blood in a free form, not bound to serum proteins. It is in reversible equilibrium with the bound hormone and represents the diffusible fraction of the hormone capable of traversing cellular membranes to exert its effects on body tissues.

The normal adult fT3 reference value is 0.25–0.65 ng/dl (3.8–10 nmol/l). fT3 measures the very tiny amount of unbound, circulating T3.

Reverse T3 is principally a product of T4 degradation in peripheral tissues. It is also secreted by the thyroid gland, though these amounts are practically insignificant. The normal range in adult serum for rT3 is 14–30 ng/dl (0.22–0.46 nmol/l), although varying values have been reported.

As with all pituitary hormones, TSH is secreted pulsatedly and has a circadian rhythm. Serum TSH concentrations are highest in the evening at 23 hours, during the first hours of sleep. Serum TSH values vary with age changes, though the normal range is approximately 0.5–4.5 mU/l. In 2003, the American Association of Clinical Endocrinologists narrowed the range of serum TSH to 0.3–3.0 mU/l.

Tg is the principal iodoprotein of the thyroid gland, produced by normal thyroid tissue. As stated above, after synthesis Tg is transported and stored in the follicular lumen, with just a fraction released into circulation. Tg concentration in serum of normal adults ranges from less than 1 ng/ml to 25 ng/ml (1.5–38 pmol/l), with mean levels of 5–10 ng/ml. Values are slightly higher in females than in males. Pituitary TSH regulates Tg secretion since serum Tg is in positive correlation with TSH. Hence, elevated serum Tg reflects increased secretory activity by thyroid gland stimulation or thyroid tissue damage, whereas Tg values below or at detectability level indicate a paucity of thyroid tissue or suppressed activity [63].

## Parathyroid glands and parathyroid hormone synthesis

Parathyroid glands are very small glands of the endocrine system, normally weighing approximately 25 g, and typically located to the side and behind the thyroid gland [1].

The parathyroid glands elaborate a peptide hormone, the parathyroid hormone (PTH), whose primary role is to prevent and/or reverse acute hypocalcaemia. The PTH achieves this by mobilizing calcium from stores in bone, stimulating renal calcium reabsorption and promoting 1,25-dihydroxyvitamin $D_3$ production to drive intestinal calcium absorption. To prevent uncontrolled elevations in plasma calcium concentration in response to PTH, a molecular feedback mechanism mediated by extracellular calcium ion concentration suppresses PTH secretion from the gland's cells. In addition to providing acute control of PTH secretion from both newly formed secretory vesicles and stored secretory granules, the calcium-mediated feedback mechanism also suppresses PTH gene transcription and cell proliferation [1,19].

Human PTH is synthesized as a large polypeptide (pre-proPTH) containing 115 amino acids that undergoes two successive proteolytic cleavages to yield 84 amino acid PTH, the main form of the hormone stored and secreted by the parathyroid glands. PTH metabolization occurs in the liver, releasing various amino-truncated fragments into the bloodstream, generically called C-terminal PTH fragments because they have kept the carboxyl-terminal part of the native PTH molecule. These fragments are cleared from the circulation through the kidney and have a much longer half-life than the 84 amino acid PTH. A circadian rhythm for PTH exists, showing a nocturnal acrophase, a mid-morning nadir and a smaller afternoon peak [10,66].

PTH clinical reference ranges are usually established in vitamin D repleted subjects with a normal renal function and depend on the used assay. Third-generation assays report reference values ranging from 5 ng/l to 39 ng/l [10,66–68,74].

## Effects of generalized and localized trauma on thyroid hormone concentrations: Clinical experience

The effects of severe generalized trauma and surgical trauma on the levels of total and free T4 and T3 and rT3 have been evaluated in the clinical setting. Significant, rapid decreases in serum T3 after trauma onset, within 24 hours of injury and in the postoperative period were noted [2,3,13,53,77].

Penetrating and blunt trauma to the anterior neck region causing thyroid gland injury have been extensively reported in clinical literature. Cases of penetrating or blunt neck trauma causing thyroid storm due to acini rupture and massive thyroid hormone liberation into the bloodstream have been described occasionally in patients with previously diagnosed or undiagnosed hyperthyroidism [9,20,21,24,29,30,40,44,59,73,75,81].

Situations of thyroid storm following local pressure to the neck caused by strangulation or suicide attempts by hanging have been exceptionally observed. Ramírez et al. [56] reported the case of a 37-year-old woman, without pre-existing thyroid disease, who was admitted to hospital after being assaulted. At the scene, she was found with a tourniquet tied around her neck. At admission, physical examination revealed a circumferential cervical contusion along with cervicofacial ecchymoses and petechiae. Plasma thyroid function tests showed TSH concentration below 0.01 mU/l (normal range 0.5–4.5 mU/l) and T4 concentration at 19.3 μg/dl (normal range 5.0–12.0 μg/dl). TSH concentration before strangulation was retrospectively determined and was normal, thus supporting the diagnosis of thyroid storm induced by local pressure to the neck in a previously euthyroid patient.

Shrum at al. [64] reported the case of a 19-year-old woman with no family history of autoimmune disorders or hyperthyroid conditions who was admitted to the intensive care unit following a suicide attempt by hanging (ligature around the neck prepared with a scarf). Laboratory tests obtained at presentation to the emergency department revealed TSH concentration below 0.01 mU/l. fT3 and fT4 were initially within normal limits and slightly elevated respectively. Specific TSH receptor antibodies were within normal clinical limits. Alternative causes of hyperthyroidism were investigated and none was found, thus corroborating the hypothesis of thyroid storm induced by hanging in a previously euthyroid patient.

## Postmortem behaviour of TSH, thyroid hormones and parathyroid hormone

Laboratory investigations focusing on the postmortem stability of thyroid hormones, TSH and PTH in biological fluids collected at autopsy have been performed by some research teams in the past with variable results.

Postmortem serum TSH levels were found to show minor variations compared to antemortem levels and remain fairly constant for at least 24 hours after death, making its postmortem determination useful for the diagnosis of thyroid dysfunctions. Measurable TSH levels were also detected in vitreous humour, though not systematically and with no relation to the postmortem serum levels.

Postmortem serum T4 levels were demonstrated to decline at an erratic, individual rate after death, causing postmortem concentrations to be in the range of hypothyroid individuals in some cases without thyroid disease. These findings suggested prudence in interpreting low postmortem T4 levels as evidence of hypothyroidism. Conversely, high postmortem serum levels of T4 were used by some authors to support the postmortem diagnosis of thyroid dysfunction and fatal thyrotoxicosis.

T3 levels were found to increase, decrease or remain unchanged after death with variable courses, making their interpretation difficult for antemortem thyroid status prediction in the deceased. Higher T3 levels were attributed to postmortem conversion of T4 to T3, whereas lower levels were hypothesized to be due to postmortem bacterial degradation of the molecule [8,14,16–18,27,28,34–36,45,55,65].

As emphasized by Edston et al. [28], the sampling sites for TSH and thyroid hormone determination have varied in different studies, with blood from both subclavian vessels and the heart having been used. Since postmortem diffusion from the thyroid gland might have occurred at those sites, Edston et al. [28] used blood from the femoral vein to minimize this risk. They observed that postmortem fT3 and fT4 values were fairly comparable to antemortem clinical reference values, though the upper normal limit, especially for fT4, had to be adjusted upward.

Based on their results, the authors concluded that measurements of blood fT3 and fT4 could be useful in the postmortem setting, in combination with histology and upper normal limit fT4 adjustment. Contrary to the results of former investigations, which had found T4 and T3 undetectable in vitreous humour despite their small molecular size, Edston et al. [28] found measurable vitreous fT3, fT4 and TSH levels. A positive correlation between blood and vitreous levels was seen exclusively in the case of fT4, though vitreous levels were approximately 30 per cent of blood levels. Conversely, no positive correlations were observed between blood and vitreous fT3 and TSH concentrations, leading to the conclusion that vitreous humour cannot be used to assess thyroid function.

Postmortem serum PTH concentrations were demonstrated to remain stable after death or show moderate elevations in concentration up to 17 hours after death. In addition, measurable PTH levels were detected in vitreous humour and subretinal fluids in human eye [7,17,18,55].

## Postmortem biochemical investigations and their application to the diagnosis of mechanical compression of the neck region inflicted prior to death

The possibility that thyroid and parathyroid glands, located in a small anatomical area unprotected by bone or dense musculature, might be significantly damaged following mechanical compression of the neck region and thus release Tg, thyroid hormones, calcitonin and/or PTH into the bloodstream has captivated interest from forensic pathologists in the past and led some research teams to question whether specific postmortem biochemical investigations could provide additional evidence in supporting the hypothesis of antemortem mechanical trauma to the neck. Initial observations in this line of thinking date back to the early 1970s and further reports have subsequently followed.

Tg leakage into the circulation of victims dying after mechanical force on the neck region was applied (either manual strangulation or throttling) was first reported by Yada et al. [78–80]. These authors used heart blood as specimen and precipitation-electrophoresis as an analytical technique.

Yada et al. [78–80] started from the concept that Tg is exclusively produced and stored in thyroid gland follicles and that Tg leakage into the circulation is unlikely to occur under normal conditions, whereas this typically characterizes certain disorders of the thyroid gland. They therefore assumed that mechanical force applied to the neck area might somehow damage the normal structure of the thyroid gland (especially the upper portions of the right and left lobes as well as of the pyramidal lobe) to such an extent as to trigger the release of detectable amounts of Tg into the systemic circulation through blood and lymphatic vessels draining from the structurally impaired gland.

The authors highlighted that it should be reasonable to expect right ventricle blood to surpass left ventricle blood in the content of Tg due to its jugular vein blood. They also emphasized that a certain number of mechanical neck trauma cases with negative blood Tg were to be predicted.

Succeeding research by Katsumata et al. [43] compared Tg determination in postmortem serum from cardiac blood in victims of mechanical asphyxia (throttling, strangulation and hanging) using highly sensitive radioimmunoassay and precipitation-electrophoresis methods, the latter recommended for everyday practice. The results of this study revealed the presence of abnormally high postmortem serum Tg levels in situations of mechanical asphyxia, although some cases of hanging were characterized by normal postmortem serum Tg levels. According to the authors, this might be attributed to the possible absence of mechanical force directly applied on the thyroid gland in these cases.

Detectable levels of Tg were subsequently demonstrated in dried blood stains (using precipitation-electrophoresis method) and denatured, half-haemolysed blood (using radioimmunoassay method) by Katsumata et al. [41,42].

The use of highly sensitive enzyme-linked immunosorbent assay (ELISA) method for Tg detection in cardiac blood postmortem serum samples was later proposed by Tamaki et al. [70], who measured Tg levels in a series of forensic cases including causes of death unrelated to mechanical asphyxia as well as hanging, strangulation and throttling cases. Individuals with causes of death other than mechanical asphyxia ($n = 15$) had Tg levels ranging from 23 ng/ml to 162 ng/ml. These values were higher than those found in normal subjects but showed no relationship to postmortem intervals up to 24 hours, thus putting the hypothesis of massive postmortem Tg leakage from the thyroid gland into question. On the other hand, Tg levels ranged from 100 ng/ml (in a case of strangulation) to 13.400 ng/ml (again, in a case of strangulation) in the 14 victims of mechanical asphyxia with only two cases of strangulation with Tg levels lower than 200 ng/ml, thus allowing these cases to be clearly distinguished from those with causes of death unrelated to mechanical asphyxia.

In a subsequent study, Tamaki et al. [69] compared Tg determination in cardiac blood postmortem serum samples in victims of mechanical asphyxia ($n = 42$, including hanging, strangulation and throttling cases) and control individuals without neck injuries ($n = 36$) using highly sensitive ELISA method and radioimmunoassay method. In addition, they measured T3, T4 and TSH levels in 21 out of 42 victims of asphyxia due to external compression of the neck and 22 out of 36 individuals without neck injuries. A highly significant correlation was observed between the ELISA and radioimmunoassay methods. Individuals with causes of death other than mechanical asphyxia had Tg levels ranging from 8 ng/ml to 162 ng/ml. Values were systematically lower than 200 ng/ml and were systematically higher in right heart blood postmortem serum than left heart blood postmortem serum. Tg levels in victims of mechanical asphyxia due to neck compression were in the range 28–5900 ng/ml. Values were systematically higher in right heart blood postmortem serum than left heart blood postmortem serum. Seven cases out of 42 had Tg levels lower than 200 ng/ml. However, all 14 cases of throttling had Tg values higher than 200 ng/ml. Although mean values of T4 and TSH in asphyxia cases were higher than control cases, most individual values in both groups were within the normal range. Furthermore, T3 levels were abnormally high in most cases and widely overlapping in asphyxia cases with control cases.

Müller and co-workers made a significant contribution to investigating Tg and thyroid hormone concentrations in heart blood samples obtained from mechanical asphyxia cases (including hanging, strangulation by ligature and throttling) and control cases [49–52]. These authors observed higher Tg levels in heart blood serum samples in individuals who died by hanging (Tg levels ranging from 12 ng/ml to more than 400 ng/ml) compared to sudden death cases. Moreover, the highest Tg concentrations were measured in cases of incomplete suspension (i.e. hanging in a sitting or semi-reclining posture), possibly suggesting an influence of agony duration on heart blood Tg levels [49]. In a subsequent study, Müller et al. [50] found higher T4 concentrations in heart blood serum in cases of hanging as well as higher T3 values in heart blood serum in cases of unexpected natural death and hanging. In another report, Müller et al. [52] measured Tg concentrations in blood samples obtained from both heart ventricles in hanging, throttling and strangulation by ligature cases. Comparisons pertaining to the obtained heart blood Tg values were performed among the asphyxia subgroups themselves as well as between asphyxia subgroups and control cases (sudden deaths). The highest mean Tg concentration was observed in throttling cases (561.6 ng/ml). Although the highest Tg values were measured in throttling and hanging cases (853 ng/ml and 892 ng/ml respectively), value ranges in cases of hanging (4–892 ng/ml) and strangulation by ligature (2–628 ng/ml) were larger than in throttling cases (252–853 ng/ml). Furthermore, statistically significant differences were found between throttling and strangulation by ligature as well as between throttling and hanging. Müller et al. [52] emphasized that, although potentially contributory to the elucidation of antemortem mechanical asphyxia, postmortem Tg values should not be used individually to confirm a vital reaction diagnosis in cases of suspected external neck compression but always in association with morphological findings obtained from autopsy.

Increased fT3 (mean value 7.27 pg/ml) and Tg (mean value 268.46 ng/ml) levels in cases of hanging ($n = 24$) compared to control cases (25 sudden deaths, fT3 mean value 4.72 pg/ml and Tg mean value 71.54 ng/ml) were found by Senol et al. [62] in cardiac serum specimens obtained by mixing blood sampled from the right and left chambers of the heart. Tg concentrations in hanging cases were higher than in control cases, but there was no statistically significant difference (using chi-square statistical assessment) between the two groups, nor were there any pertaining to amylase, PHT, calcitonin, fT4 or FSH heart blood levels (the two latter indeed useful for thyroid dysfunction evaluation). Conversely, the difference concerning heart blood fT3 concentrations in asphyxia and control cases was found to be statistically significant (using chi-square statistical assessment). It is worth noting that Mann–Whitney U test assessment revealed statistically significant differences between hanging and control cases concerning not only fT3 values but also Tg levels. Lastly, evaluation of cases characterized by simultaneous increases in Tg and fT3 concentrations revealed statistically significant differences (using chi-square statistical assessment) between asphyxia and control cases, thus suggesting high levels of both markers as potential vital reaction indicators in hanging cases.

Recent investigations made by Ishikawa et al. [39] and Hayakawa et al. [31,32] revealed measurable TSH levels in cerebrospinal fluid (using an electrochemiluminescence immunoassay) in a series of forensic autopsy cases including asphyxia. Increased Tg levels (over 200 ng/ml, using an electrochemical luminescence immunoassay) were noted in both right and left heart blood samples collected separately in forensic autopsy cases with causes of death unrelated to external neck compression or thyroid disease. Values in right heart blood samples were significantly higher than left heart blood samples, possibly suggesting Tg leakage from the thyroid gland into circulation after death and increases in Tg concentrations in heart blood due to postmortem changes, thereby making Tg levels inappropriate for diagnostic purposes in the presence of large differences between right and left heart blood Tg values. Blood Tg concentrations in forensic autopsy cases without findings presumed to increase Tg levels were significantly higher in right heart blood samples (compared to left), external iliac venous blood samples (compared to external iliac arterial ones), right heart blood samples (compared to external iliac venous blood samples) and left heart blood samples (compared to external iliac arterial blood samples). This possibly indicates that Tg is more readily diffused to the venous system than the arterial system after death (due to higher blood volume retained by the venous system postmortem) and to heart blood compared to peripheral blood (due to heart to thyroid gland proximity).

Based on these results, Hayakawa et al. [32] concluded that postmortem changes and heart to thyroid gland proximity may significantly influence Tg concentrations in blood samples obtained from different sites, leading to the conclusions that peripheral arterial blood situated far from the thyroid gland is a more appropriate sample for forensic diagnosis.

## Conclusion

As illustrated in this chapter, despite a certain number of significant observations showing increases in blood Tg levels in forensic cases of hanging, strangulation by ligature and throttling, a general consensus exists regarding the fact that it is not recommended to use postmortem biochemical results to support the hypothesis of mechanical force applied to the neck prior to death in the absence of consistent macroscopic and microscopic data. The latter are necessary to confirm the presence of thyroid injuries and rule out the existence of thyroid disease.

In addition, some authors have hypothesized and demonstrated that results obtained can be significantly influenced by the blood sample site chosen since diffusion of thyroid hormones and Tg from the thyroid gland may occur at cardiac levels postmortem.

Yet again, some authors have recommended the use of femoral vein blood samples concerning postmortem biochemistry on peripheral blood specimens, while others have concluded that peripheral arterial blood situated distant from the thyroid gland is a more appropriate specimen for diagnostic purposes since higher quantities of blood remain in venous vessels after death.

Prompt systematic peripheral blood sample collection and analysis is the hallmark recommendation for biochemical investigations of Tg and thyroid hormones in the forensic setting. These should be carried out as soon as possible after body admission to the morgue. Lastly, and most importantly, obtained laboratory results must be combined with morphological findings to provide more thorough further information on the deceased's thyroid function prior to death.

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# 17 Intoxication as a Risk Factor

*Henrik Druid*

## General aspects

Given the increasing use and abuse of pharmaceutical drugs, illicit drugs and new psychoactive substances not yet classified, today it is likely that a toxicological analysis of blood from an asphyxiated victim will reveal one or more such substances. In addition, alcohol remains a common finding in forensic pathology casework. This means that the toxicology will often have to be taken into account when different alternatives for the cause(s) and manner of death are considered. Since many forms of asphyxia are not incompatible with survival but facilitated by various factors, it is important for the forensic pathologist to review all findings before making the diagnosis. Whereas the toxicological results in complete hangings, or in cases with severe chest compression with unambiguous findings and circumstances, most often do not make any difference to the investigator, the alcohol and drug levels will have to be carefully considered in most other cases of fatal asphyxia.

## Toxicology in accidental asphyxiations

Bolus deaths are regularly encountered by forensic pathologists. The victims are usually either chronic alcoholics or elderly with dementia, or patients with swallowing problems due to neurological disease. In the case of alcoholics, the typical finding is a large piece of food, voraciously swallowed under the influence of alcohol, and sometimes the blood alcohol level is very high. In a study of 78 non-hospitalized persons choking on a bolus of food, one-third were under the influence of alcohol [25]. Although the mechanism underlying bolus death is still not completely elucidated, a vasovagal reflex with cardio-inhibitory signalling is one of the most likely alternatives. The normal counterbalance of this overactivation of the parasympathetic system may then fail at significant blood alcohol levels. Due to a reduced muscle strength, subjects severely inebriated by alcohol will also have problems in efficient coughing. Several of the drugs mentioned below as possible facilitating factors in positional asphyxia may also be considered as contributing to a fatal outcome in bolus emergencies.

Aspiration, particularly of gastric contents, is a very common finding at autopsy, but its significance for the cause of death is highly variable. Pathologists may overestimate the importance of an impressive aspiration, where the airways actually might not be completely blocked, whereas significant obstruction of the smaller bronchi may go undetected if the examination of the lungs is not performed accurately. Aspiration should be considered as the main or contributory cause of death only when the small bronchi all the way to the finest branches in the lung parenchyma on both sides are blocked by chyme, blood or other material [18]. Even so, agonal aspiration is very common and may in most instances be just an incidental finding. Acute pulmonary oedema and spots with discolouration on the cut surface of the lungs may be signs supporting the notion that the aspiration was vital and significant. In the latter case, microscopic examination may reveal evidence of inflammatory response with accumulation of neutrophils, suggesting that some time has elapsed between the aspiration and death. If the person was under the influence of alcohol and drugs at the time of the aspiration, then the levels might have dropped until death ensued. On the other hand, blood drug levels may increase due to postmortem redistribution if the inhaled vomits contain high amounts of alcohol or drugs. Hilberg et al. [15] administered amitriptyline to rats via a gastric tube 10 minutes postmortem and found significant levels of the drug in heart blood and in inferior vena cava blood, whereas no or minimal levels of amitriptyline were found in such samples in rats where the trachea had been ligated. Hence, aspirates containing drugs may represent an important source of artefactually elevated levels of drugs.

Aspiration of gastric contents is a very common finding in fatal intoxications. This implies that the significance of aspiration, and the possibility of postmortem redistribution, have to be taken into account when determining the cause of death [30]. However, in most cases it is very likely that the person would have died from the toxic effect of the drugs even without the final vomiting. Further, postmortem redistribution from aspirated material in the airways is less likely to affect peripheral venous blood, particularly if appropriately collected [48].

The increased risk for drowning when under the influence of alcohol has long been pointed out by authorities in many countries in order to prevent accidental deaths, particularly during the summer months in temperate climate zone countries (see Table 17.1). In a Swedish study reviewing all drownings from 1992 to 2009 [1], the homicidal, suicidal and undetermined cases were evenly distributed across the months, whereas the accidental drownings formed a conspicuous peak during June–August (Figure 17.1). Forty-four per cent of the accidental drowning victims tested positive for alcohol. In Finland, all drowning deaths between 1970 and 2000 were examined with regard to findings and circumstances [20]. More than 70 per cent of non-boating victims had a blood alcohol concentration (BAC) over 50 mg/dl. In boating-related accidents, the percentage was even higher. A similar study, covering the years 2000–2009, reported a slightly lower proportion, 62 per cent, of BAC over 50 mg/dl for non-boating drownings [27]. In this study, psychotropic drugs were also analysed: 20 per cent of the non-boating drowning victims had psychotropic drugs in their blood along with BAC more than 50 mg/dl and an additional 9 per cent of them had psychotropic drugs with lower BAC or a negative alcohol result. The authors also graded the significance of these drugs and concluded that about 16 per cent of the non-boating victims were significantly influenced by the detected psychotropic drugs at the time for the accident.

Another interesting observation in that study was that the proportion of alcohol-positive cases showed a gradual decrease from 80 per cent among those aged 50–54 years to less than 20 per cent for those aged 80–84 years. This panorama probably reflects the increased risk for a fatal outcome in a drowning situation among elderly, regardless of alcohol intake or not. In a recent study from Hungary, 85 per cent of the male drowning victims and 52 per cent of the female victims had consumed alcohol [32]. Lower proportions, of 44 per cent, 41 per cent, 40 per cent and 33 per cent, of alcohol-associated drownings were reported from New York State [4], California [46], Australia [28] and Alaska [42] respectively. These differences might be partly explained by cultural factors and geographical conditions, but the figures are still high enough to justify continued efforts to reduce these untimely deaths. The exact blood concentration at which alcohol significantly impacts the chances of surviving a near-drowning situation cannot be determined, but the small changes in heart rate, respiratory rate and heat loss observed in the classic experimental study by Keatinge and Evans [17] do not support significant effects of alcohol at blood levels below 100 mg/dl. Further, alcohol at a BAC of approximately 100 mg/dl did not produce any significantly different physiological response to the initial cold shock among healthy subjects [12]. It is therefore reasonable to assume that the association between alcohol and drowning rather is a result of the deleterious effect of alcohol on judgement, awareness and coordination than on CNS depression.

Bathtub drownings constitute an interpretational problem similar to aspiration of gastric contents in intoxication subjects. The difference is, of course, that an aspiration can produce a prolonged process of impaired respiration and result in a delayed death whereas any drowning will go fairly quickly. The problem, however, is that a person who has taken drugs may commit suicide in the bathtub by intentionally inhaling water or may just wait until reaching a state of reduced consciousness when the drowning will be a more or less passive process. The latter may also be true for unintentional drownings among intoxicated persons. Because there is as yet no published compilation of drug levels found in bathtub victims that could be used as a reference, it seems reasonable to use particular caution when evaluating such cases.

Positional asphyxia implies that a person has assumed an unnatural position which compromises their respiration. In some cases, the person is trapped in such a position from which it is difficult to escape, but more commonly the victim has sustained injuries during a fall, or is intoxicated by alcohol or drugs, reducing their ability to change position. In general, alcohol intake is associated with increased risk for accidental deaths, including those caused by fatal asphyxia. In addition to the risk for falls and adopting

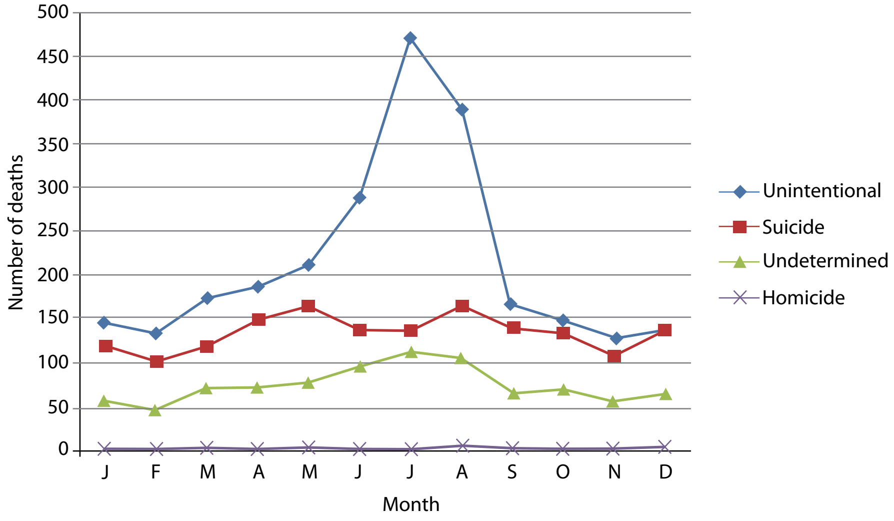

**Figure 17.1** Number of drowning deaths in relation to months and manner of death in Sweden from 1992 to 2009 ($n = 5125$) (modified from Ahlm K, Saveman BI, Bjornstig U. *BMC Public Health* 2013;**13**:216).

an abnormal position, the influence of alcohol may also contribute physiologically to the asphyxia by reducing the capacity to maintain free airways. In experimental settings, even moderate amounts of alcohol have been shown to reduce the tension of the genioglossal muscle complex in awake subjects, increasing the risk that the tongue will fall back and block the pharynx as the person fails to maintain their alertness [19]. Epidemiological studies have shown an association between alcohol and sleep apnoea [47] and, experimentally, during sleep following moderate alcohol intake (producing an average BAC of 0.07 mg/dl), both nasal and pharyngeal resistance increased significantly in subjects with or without pre-existing obstructive sleep apnoea disorder [34,35]. These results were confirmed in other studies, in which the number of sleep apnoea events exceeded the normal limit after similar moderate alcohol intake [24,39]. Thus, there is ample evidence to support the notion that alcohol promotes asphyxiation in cases where the victim has adopted an abnormal position compromising the process of respiration. In addition, the general respiratory depressant effect of alcohol, as well as the reduced strength, and in many cases, the effect of alcohol on judgement and coordination, may contribute to a fatal outcome. It is therefore often justified to consider alcohol as a significant contributory factor in positional asphyxia deaths.

Pharmaceutical drugs may also be important in positional asphyxia by attenuating the genioglossal tone and/or by affecting other systems important for airway patency. The pedunculopontine tegmental nucleus has been identified as an important control centre for the regulation of upper airway muscle tone. In a study in rats, Saponjic et al. [37] showed that N-methyl-D-aspartate (NMDA) and NMDA antagonists introduced specifically into this nucleus could activate and block, respectively, the genioglossus muscle tone. In addition to these electromyogram recordings, they showed that the same treatments to some extent also modulated the breathing pattern. Similar experiments focusing on the role of the retrotrapezoid nucleus and the Kölliker-Fuse nucleus in the brain stem on the respiratory pattern and the tone of the upper airway muscles were performed by Silva et al. [40]. They found that NMDA injection into the retrotrapezoid nucleus of NMDA increased diaphragm and genioglossus muscle activities and that this response to NMDA stimulation or by hypercapnia was reduced after injection of the $\gamma$-aminobutyric acid GABAA agonist muscimol into the Kölliker-Fuse nucleus. These publications and other experimental studies provide support for the observation that NMDA antagonists, such as tramadol, methadone, ketamine and dextrometorphane, as well as GABA agonists (e.g. alcohols, benzodiazepines, chlormezanone and carbamates) can compromise the breathing at variable blood concentrations, depending on their receptor specificity, affinity and efficacy. In addition, certain drugs, such as the antipsychotic drug clozapine, can also cause hypersalivation [31]. A person who has adopted an abnormal, yet supine position, and is either comatose or immobilized, may aspirate saliva, and if a drug-induced hypersalivation is present, the amounts may be sufficient to compromise the airway flow and contribute to the asphyxia.

Alcohol, sedatives, hypnotics and certain illicit drugs share a general central nervous system (CNS) depressant effect, which may contribute to asphyxia in subjects who have adopted an abnormal position with reduced airway flow. However, it is a challenge to estimate the respiratory depressant effect only by reviewing the postmortem blood drug concentrations. This also applies to what has been said about the particular drugs' negative effect on the upper airway resistance. In order to be considered significant, the drug concentrations should clearly exceed those that can be seen in deaths where the victim obviously was not incapacitated by drugs [10]. Further, the general notion that the combination of alcohol and CNS depressant drugs, or combination of several CNS depressant drugs, will increase the toxicity is supported by epidemiological studies, whereas there is a lack of well-designed experimental studies focusing on the pharmacodynamic interactions. Hence, a humble attitude is warranted when assessing the impact on blood drug levels for the fatal outcome in cases of positional asphyxia.

A particular form of positional asphyxia is when the body is lying head-down, or even hanging upside down. A number of case reports have described such cases and the circumstances are typically unique to each case. In a review of existing publications Sauvageau et al. [38] reported that 6 of 16 victims were intoxicated by alcohol, although in a few of their cases no toxicology was carried out. In more recent publications, the pattern is similar; about half of the victims were seemingly influenced by alcohol or drugs at the time of the incident. The most likely mechanisms suggested to lead to a fatal outcome – exhaustion of the blood pressure regulation and failure of diaphragm persistence – are both likely to be potentiated by alcohol and drug influence. Given the importance of diaphragm exhaustion for the fatal outcome, drugs that reduce the neural signalling to the diaphragm are likely to facilitate the process.

Fatal asphyxiation caused by chest compression, often referred to as traumatic asphyxia, will in most cases lead to death fairly rapidly. The petechiae typically appearing on the head, neck and shoulders are not a proof of a long agony but may be produced within minutes. The possible influence of alcohol or drugs will therefore usually not make much difference to the fatal outcome. In a review of 35 cases of traumatic asphyxia, Sklar et al. [41] reported that 5/5 survivors had no alcohol in their blood, whereas 7/30 fatal cases had blood alcohol levels exceeding 0.1 g/l. However, the presence of alcohol or drugs in such deaths may rather be associated with an increased risk for the event as such by contributing to, for example, incautious operation of a heavy machine, or poor driving ability, regarding traumatic asphyxia due to motor vehicle accidents.

Chest compression may also be a factor in sudden deaths associated with subdual of violent persons by the police, guards, paramedics or healthcare professionals. The actions taken may involve both restraint and chest compression. In many cases, positional asphyxia has also been suggested to have played an important role in these deaths. A large number of violent persons resisting arrest or emergency care are under the influence of alcohol, yet sudden deaths during apprehension and subdual of drunk subjects is rare. It was observed relatively early that many of the sudden deaths in custody comprised subjects with an altered mental status, and Wetli and Fishbain in 1985 [45] linked many of these deaths with cocaine-induced psychosis, today called 'excited delirium'. A large number of case reports and case series were subsequently published. In parallel, many reports dealt with positional asphyxia as a competing cause for these in-custody deaths, and a number of experimental studies on human subjects were performed to evaluate the effects of various restraint measures and body positions on respiratory and circulatory parameters [7,8,33]. However, the limitation of these studies was that all volunteers were healthy subjects and were not under the influence of drugs. During the last two decades, a fairly large number of papers have been published reporting on sudden in-custody deaths associated with excited delirium. Most commonly, the excited delirium cases have been associated with cocaine abuse, but the syndrome has also been described in association with use of methamphetamine, MDMA, alpha-PVP, methylone and ephedrine [22,23,29]. Virtually all drugs associated with excited delirium are psychostimulants that result in an increase in extracellular dopamine levels in critical brain regions. The main pathophysiological explanation for the clinical symptoms is a dopaminergic overdrive, due to a failure of the affected person to upregulate the dopamine transporter protein [22], although a general depression of the cholinergic system in the brain probably contributes to the delirious state. The severity of this acute condition is well recognized today. In the acute phase, death may be caused by cardiac ischaemia due to a tremendous increase in energy demand during extraordinary exertion and sympatico-mimetic overdrive. Hyperthermia is almost always recorded, and may cause death after a while, if unrecognized, or left uncontrolled. Subjects surviving these phases may die later due to massive rhabdomyolysis. The problem is that many of these deaths occur early and unexpectedly during a violent fight, and hence before any medical examination is possible. Postmortem examination of the dopamine transporter protein in ventral striatum and expression of the *HSPA1B* gene and its product HSP70 in the temporal cortex has been suggested as with markers of excited delirium [23]. However, even if this condition has been observed to cause death in the absence of any intervention [36], in most cases restraints, chest compression and abnormal body position are factors that the pathologist needs to consider when evaluating the case.

## Toxicology in suicidal asphyxiations

In several countries, plastic bags have been labelled with warning text that the bags may be dangerous to small children, yet such cases are rarely reported. In a retrospective review spanning 20 years, no unintentional deaths due to plastic bag suffocation were identified in the Milan region [9]. On the other hand, plastic bags as a means for committing suicide has been observed for decades, and the method has even been recommended in books on suicide methods and assisted suicide as well as on web pages, possibly affecting the incidence. In most cases, the plastic bag is simply tightened around the neck, but in a proportion of the cases, gas is introduced through a tube into the bag. Most often such arrangements involve a connection to a helium tube [3,44], but there are also reports on the use of nitrogen [21], propane and butane [9] and even ether or chloroform [49]. In the report on chloroform deaths, one subject had chewed a piece of cotton soaked with chloroform, and another applied impregnated surgical masks around the mouth and nose. Toxicological analysis may reveal the presence of these volatiles, but the interpretation is not straightforward, since the concentrations of each of them is difficult to interpret. However, the mere presence of such volatiles in blood or brain tissue samples will in most cases suggest that the person had been inhaling a substantial amount, and even serve as a support for suicide rather than accidental suffocation.

Fatal suffocation using a plastic bag does not require a weakened state conveyed by sedative drugs or a medical illness. The proportion of cases with positive toxicology is variable. In a retrospective review in Australia, Byard et al. [6] reported that toxic or fatal concentrations of prescription medications were found in 17 of 45 cases. In a study from Ontario, Canada, 92 per cent of the victims tested were positive for drugs; the most common drugs were benzodiazepines, diphenhydramine and antidepressants with diphenhydramine being the most common drug found in elevated levels in blood [5]. In other compilations, lower proportions of drug detections are reported, and in several case reports, no drugs or alcohol were found. Given the fairly short time that suffocation in a plastic bag can result in death, it is not surprising that fatal levels of drugs are uncommon in suicidal settings. If the arrangement obviously is completely airtight, a very high concentration of drugs that can kill should raise concern as to whether the manner of death really is suicide, since the person should shortly before death have managed to arrange, put on and adapted the bag around the head and neck. In some cases, however, the bag is not completely tightened, allowing exchange of air, implying a prolonged process during which blood drug concentrations may build up.

Drowning is fairly common as a suicidal method and, in most case series, half of the victims are female. In a large retrospective study, 55 per cent of the female drowning victims had committed suicide as compared to 21 per cent of the males [1]. In the same study, 24 per cent of the suicide victims tested positive for alcohol, and 69 per cent had one or more drugs in their blood. In a review of studies on drowning in different countries, the presence of alcohol and drugs in the blood of suicidal drownings varied considerably; in several studies, alcohol was more frequently detected than pharmaceutical drugs [14]. According to the same review article, illicit drugs were uncommonly found in suicidal drownings. The question of whether alcohol and/or drugs may have contributed to death is often difficult to answer. Individuals who wish to commit suicide in water may actively go under the surface and inhale water following an intake of alcohol and drugs without being incapacitated, whereas others may enter a bathtub and just await the effect of a drug. In the latter situation, the subject may either drown when drowsy and incapacitated due to the intake, or they might actually die of poisoning and then slide down and submerse after death. Alcohol as well as any drug that causes severe sedation should be considered in both of these respects. In a study of bathtub deaths in Maryland, 8 out of 66 were suicides, and in all 8 cases, drugs were detected in the blood (diphenhydramine, zolpidem, alprazolam or morphine) [26]. Some of the victims had also consumed alcohol before their demise.

Hanging is one of the most common suicide methods worldwide, particularly frequent among males. In a study of 4551 hanging suicides, all of the top ten substances detected in the blood were drugs that are also common in fatal intoxications [16]. Among the 30 per cent of the victims positive for alcohol, the average BAC was 1.39 g/l. In a proportion of cases, the alcohol concentration exceeded 3.0 g/l, implying that these individuals obviously still were able to hang themselves. Even if a few of the hangings were arranged so that the person could more or less passively slide into the noose and asphyxiate, most of the cases were classical hangings requiring some kind of physical effort to complete the task, despite high concentration of alcohol and/or sedatives, hypnotics or antidepressants. Hence, studies of toxicological results in hangings and in other forms of deaths where incapacitation by drugs can be excluded are important sources of information to understand what levels can be tolerated in other forms of possible asphyxiations that are not so definitely associated with a fatal outcome [10,11].

## Toxicology in homicidal asphyxiations

In general, the impact of toxicological influence on various types of homicides (excluding poisonings) is often difficult to estimate, in particular because the external force leading to death is often case-specific and does not follow templates that are typical in accidental and particularly suicidal deaths. This means that the levels of alcohol and drugs found in victims of homicidal strangulations, drownings and other forms of asphyxia deaths usually are difficult to translate into a particular degree of incapacitation. Having said that, intoxication is obviously an important risk factor in homicidal strangulations. In a study focusing on offenders of 19 strangulations, most offenders were influenced by alcohol or drugs, but also at least 11 of the victims were intoxicated by alcohol and/or drugs [13]. Lower, yet significant, proportions were found among strangulation victims in Johannesburg, South Africa: 33 per cent of the male victims and 18 per cent of the female victims had blood alcohol levels above the legal threshold of 0.5 g/l, supporting the notion that male subjects are more vulnerable to this form of violence when in a weakened state, such as by high alcohol levels [43]. In a study on drownings in Sweden, 37 per cent of the homicidal drowning victims were influenced by alcohol, and this figure increases to 53 per cent when excluding victims that were under-aged [1], supporting the presumption that intoxication also facilitates this mode of homicide. Despite the vast number of publications on asphyxia deaths, the studies dealing with homicides are not large enough to allow for identification of specific patterns regarding intoxications in these victims. In a comprehensive review of drug-facilitated sexual assault, alcohol intoxication – most often due to voluntary consumption – was found to be the most common finding and that drug-spiking was rare [2]. This pattern is most likely true also for homicidal asphyxiations.

## Asphyxia or intoxication?

In summary, the impact of intoxication on asphyxial deaths is often difficult to estimate. The most reliable information, including some experimental data, derives from drownings and near-drownings, but not all those data can be translated to other forms of asphyxia. Although the physiological

**Table 17.1 Blood alcohol concentrations (BAC; mg/dl) among accidental drowning victims 15–64 years old ($n = 472$)**

| BAC (mg/dl) | *n* | % |
|---|---|---|
| 0 | 103 | 21.8 |
| 10–49 | 15 | 3.2 |
| 50–99 | 16 | 3.4 |
| 100–149 | 39 | 8.3 |
| 150–199 | 77 | 16.3 |
| 200–249 | 96 | 20.3 |
| 250–299 | 73 | 15.5 |
| 300–349 | 41 | 8.7 |
| 350–399 | 10 | 2.1 |
| ≥400 | 2 | 0.4 |

*Source:* Lunetta P, Smith GS, Penttila A, Sajantila A. Unintentional drowning in Finland 1970-2000: a population-based study. *Int J Epidemiol.* 2004;**33**(5):1053–1063.

effects of most drugs are well characterized, it is often difficult to estimate the effect on, for example, respiration from postmortem drug concentrations. In many instances, the question might not be limited to the possible influence by drugs for an asphyxial death, but also include the question about the cause of death, since intoxication per se may sometimes be an alternative cause of death. A careful review of the toxicological results – including consideration of the actual effects that the drug, at the levels found, may have on, for example, the respiratory system – is important in order to reach sound conclusions. Some drugs with action on the heart may cause death by producing a fatal arrhythmia but may not have any influence at all on the asphyxiation. On the other hand, a large number of sedative and hypnotic drugs that are commonly seen in deaths due to asphyxia do share central depressant effects, which may contribute to the fatal outcome. In obscure cases, such as positional asphyxia, particular attention should be paid to the effects that detected drugs may or may not have on the muscles involved in the maintenance of free airways and/or the respiratory muscles.

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# Section 5: General Remarks: Accident/Suicide/Homicide

Homicide is defined as the act of one human killing another. Homicides can be divided into many overlapping legal categories such as murder, manslaughter, justifiable homicide, killing in war, euthanasia and capital punishment (for details see Chapters 3–5).

Suicide is an act of intentionally causing one's own death.

An accident, also known as an unintentional injury, is an undesirable, incidental and unplanned event that could have been prevented had circumstances leading up to the accident been recognized and acted upon prior to its occurrence.

Statistical aspects of homicides, suicides and accidental deaths have already been discussed in Chapters 3–5. This section presents comparatively rare cases.

# 18 Homicide

*Burkhard Madea, Frank Musshoff and Peter Schmidt*

## 18.1 Fall downstairs: Accident, homicide or natural death?

### *Burkhard Madea and Frank Musshoff*

In fatal falls downstairs, men aged between 50 and 60 years are typically involved; women may be a bit younger. Falls are generally due either to alcoholization or to pre-existing, especially cardiovascular, diseases. Cause of death is normally a severe craniocerebral trauma with intracranial bleeding and cerebral contusions. Usually there are injuries to different sites of the body besides the head, with concomitant injuries found on the trunk and extremities [50,72–74,112,132].

The case described in this first section of the chapter seemed, on first impression, to be a fatal fall downstairs. The police investigations, prosecution and trial that followed raised very complicated issues. Here we will address just the medical and toxicological aspects of the case. Of course, in order to make its decision, the court was presented with much more evidence (e.g. of an extramarital affair and motive). The evaluation of that evidence lies outside our expertise, however, and will not be discussed here. The report focuses on morphological findings in fatal falls, toxicological aspects of succinylcholine (SUX) administration, and further questions which arose during the trial.

### Case history

A 34-year-old woman was found unresponsive in the early morning by an emergency physician and paramedic rescue team at the bottom of a stairway (Figure 18.1), indicating on first impression a fatal fall downstairs.

Prolonged cardiopulmonary resuscitation (CPR) ended unsuccessfully. The husband, who worked as an anaesthesiologist, reported to the emergency physician that he had found his wife unconscious and looking pale shortly after he had returned from taking his daughter to school. He reported that his wife's pulse was slow and weak and had eventually faded. Initially, he attempted CPR, including tracheal intubation and bag ventilation using his own emergency equipment. Afterwards he called the emergency services. He presented several electrocardiogram (ECG) strips to the emergency physician with the date and time annotations showing consecutive sinus bradycardia, asystole and ventricular fibrillation. Furthermore, he told the physician that his wife had had problems with her blood pressure that might have been responsible for the accident (fatal fall downstairs).

After the arrival of the rescue team, the paramedics moved the woman's body slightly in order to have more space for their resuscitation measures. During CPR by the emergency personnel, blood leaked from a laceration of the victim's right occipital region (Figure 18.2); no blood was visible at or near the head at the original position. No blood, hair or other biological traces or stains could be found anywhere on the stairs. Since the cause and manner of death were unclear, the prosecutor ordered a medicolegal autopsy, which was carried out at the same day.

### Autopsy findings

The body was of a 34-year-old woman with body length 176 cm and body weight 55 kg. In the right occipital/parietal region there was a horizontal 3 cm long laceration with only a slight haemorrhage into the subcutaneous tissue (Figure 18.3). There were abrasions of the left forehead, the shoulder, the left flank region and both knees, and signs of intensive medical care with defibrillation marks. There was acute congestion of the inner organs. There were no pre-existing diseases, no trauma to the skull, meninges and brain, and no fractures of the spine, ribs and extremity bones. Macroscopically, no anatomical cause of death was apparent. All organ weights were within the normal range for age (right lung 640 g, left lung 470 g, heart 260 g, brain 1320 g). The lungs showed only slight congestion and oedema without emphysema.

Further extensive neuropathological, histomorphological and molecular pathological investigations were carried out and also revealed no cause of death. The heart showed a regular structured myocardium without inflammatory infiltration, with no evidence of myocarditis or vasculitis. CD68 positive macrophages and CD3 positive T-lymphocytes were in the normal range. Nested PCR for enteroviruses, adenoviruses, human cytomegalovirus, Epstein–Barr virus on paraffin-embedded myocardial tissue were negative; only nested PCR for Parvovirus B19 on myocardial tissue and blood was slightly positive. However, this positive reaction could not be confirmed by *in situ* hybridization. Therefore, there was no evidence of

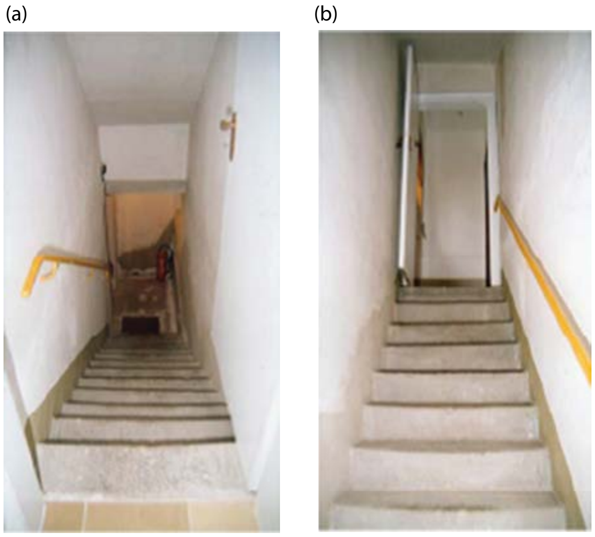

**Figure 18.1** (a),(b) View down and up the stairway where the 34-year-old woman was found.

acute or chronic myocarditis. The lung showed congestion and slight alveolar oedema, but no fat embolism.

Macroscopic and histological neuropathological investigations with different stains (haematoxylin eosin, luxol fast blue, Prussian blue, bodian, GFAP, MIB1, KP-1, HLA-DR, Tau, p53) revealed no evidence for acute traumatic brain injury or acute neurological diseases. At autopsy there were especially no coup or contrecoup lesions or traumatic haemorrhages of the meninges [64,102]. Further histology was age-related.

## Chemical–toxicological findings

Various body fluids and organ tissues were assayed for ethanol and drugs of abuse (acidic, basic and neutral organic drugs) using routine methods including immunochemical procedures and liquid–liquid as well as solid-phase extraction procedures with further analysis by high-performance liquid chromatography with diode array detection (HPLC/DAD) and gas chromatography/mass spectrometry (GC/MS). Only atropine (administered by the victim's husband) was found in the femoral blood at a concentration of 0.16 mg/l and all other tests yielded negative results.

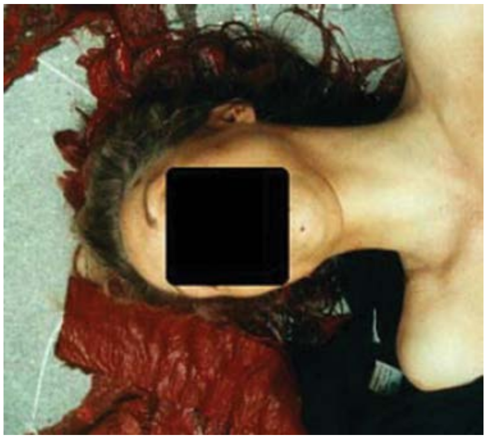

**Figure 18.2** Blood beside the head which was not apparent at the arrival of the emergency physician and the paramedic rescue team.

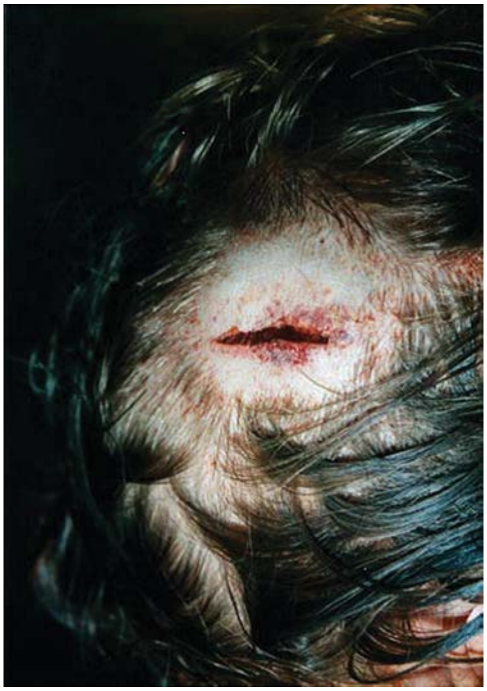

**Figure 18.3** Laceration of the right occipital region.

As documented by the police, an SUX ampoule from the husband's emergency case was missing. However, target analyses for the detection of SUX and succinylmonocholine (SMC) according to previously published procedures using thin-layer chromatography [10], GC/MS [10,59,117] and liquid chromatography/mass spectrometry (LC/MS) [12,85,113] revealed negative results.

## Further investigations

Further police investigations at the scene later in the day revealed that the husband of the deceased tried to conceal an ECG strip. When asked by the police, the husband said that the ECG strip which he had presented to the physician in the morning was of no importance. The ECG strips (Figure 18.4) were secured for further investigation.

At this point the possibility of murder rather than an accident was considered, and suspicion was raised further by a discrepancy between the initial sinus bradycardia and the stated but unsuccessful resuscitation measures, the SUX ampoule considered missing from the anaesthesiologist's emergency case, and testimony regarding the couple's seriously disturbed relationship. The anaesthesiologist

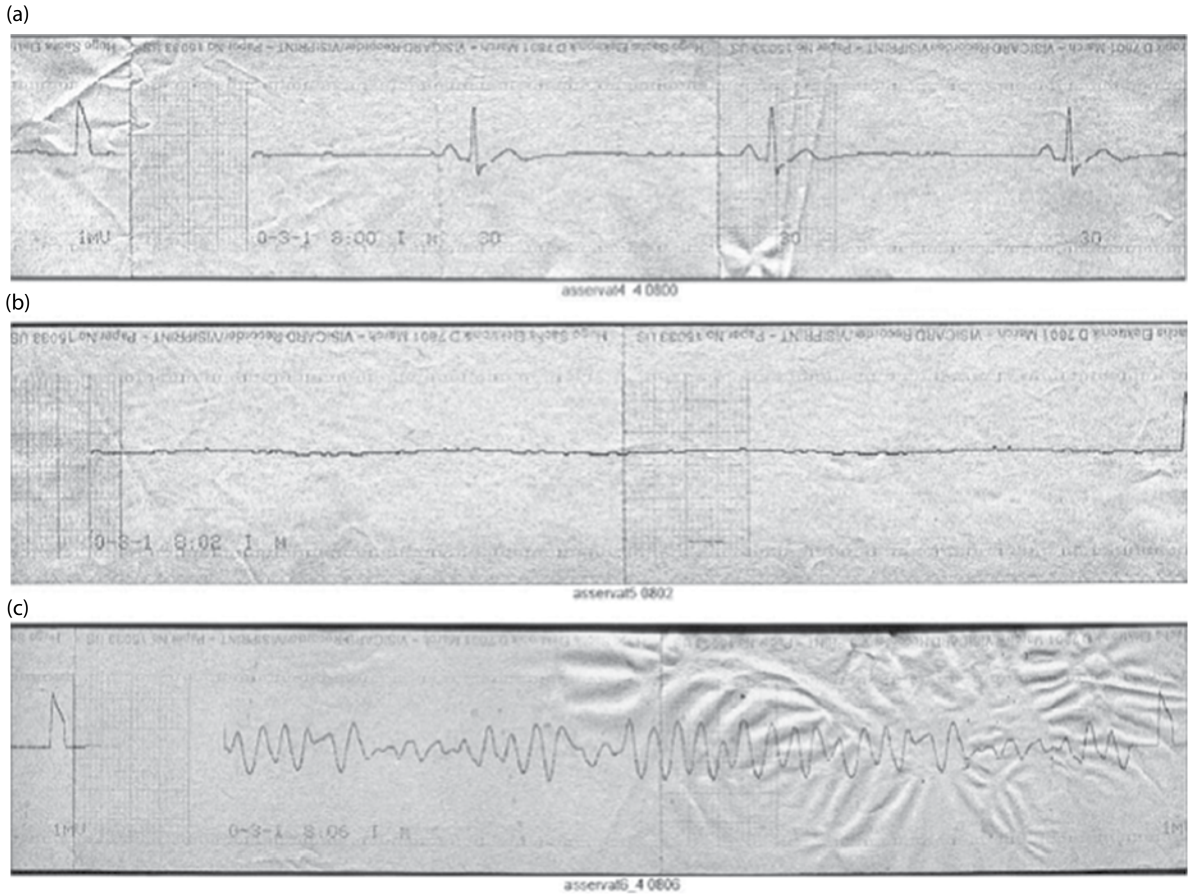

**Figure 18.4** ECG strips: (a) sinus bradycardia; (b) asystole; (c) ventricular fibrillation.

worked at a hospital far from his home, and during the week his wife lived with a person with whom she was having an affair. A planned divorce raised financial problems.

Since by morphological, molecular–pathological and toxicological investigations no cause of death was apparent and a fatal fall downstairs was ruled out because the typical autopsy findings were missing, an anaesthesiologist was asked to give a statement on the case. The expert opinion of the anaesthesiologist was based on the autopsy findings, further forensic investigations, the ECG strips, and especially the reports of the husband and the emergency doctor. According to his expert opinion:

- The woman was unresponsive during CPR by the husband and emergency doctor.
- She still had a palpable pulse.
- She was not cyanotic; therefore, a generalized oxygen deficiency was not present initially.
- According to the ECG strips, cardiac function was still preserved.

The expert anaesthesiologist came to the conclusion with virtual certainty that Mrs X did not fall down the stairs and that Dr X did not carry out resuscitation measures on his wife. In his opinion, the death of the young, healthy woman could only be explained by a deliberately induced lack of oxygen, probably caused by an SUX injection. Furthermore, the anaesthesiologist was sure to have seen signs of 'struggle' on some photographs of the deceased's house. Due to this witness statement, the husband was charged with first-degree murder by using SUX. However, the expert witness was suspected of bias and therefore objected from the court. Prior to that, he presented a further statement indicating that some ECG strips may be not authentic but faked with a simulator.

## Trial

### Faked ECG

During the subsequent trial, several questions were discussed and several experts (forensic pathologists, toxicologists, anaesthesiologists) were heard. The anaesthesiologists, some appointed by the court and some by the defence, had to investigate whether the ECG strip chart recordings, showing ventricular fibrillation and presented by the charged physician as being recorded

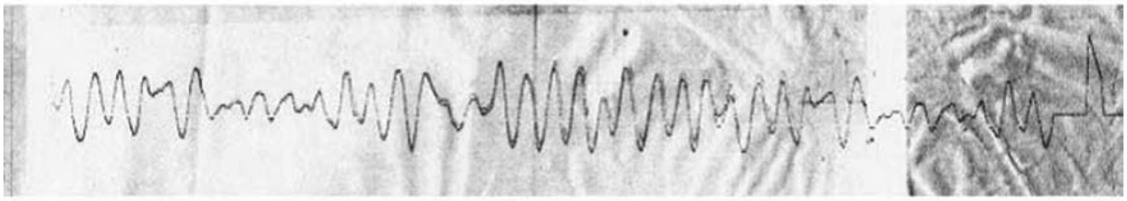

**Figure 18.5** Superimposition of the ECG-strip claimed to be recorded from his wife and an ECG strip showing cardiac fibrillation from a simulator.

from his wife during CPR, were authentic or faked with a simulator. In the trial a further anaesthesiologist was asked by the court to testify as an expert witness. According to his report, it is possible to record ECG strips identical to those secured in the present case using the ECG machine Visiprint with a simulator, especially a simulator produced by Laerdal comprising the program Heartsim2000. According to his testimony, parts of the ECG strips were undoubtedly derived from the ECG simulator and not from the deceased [128]. In cardiac arrhythmias such as ventricular fibrillation, an incidental conformity can be excluded (Figure 18.5). In addition to the anaesthesiologist, a cardiologist was heard as an expert. They both considered the probability that a period of fibrillation may show both the same periodicity and the same pattern morphology as a specific simulator's fibrillation tracing as zero, suggesting that the ECG (including date and time annotations) was faked [128]. The faked ECG was, of course, a further hint that the husband might have been involved in the death of his wife. Obviously, the faked ECG strips were shown to the emergency doctor to demonstrate cardiac arrhythmias including asystole and fibrillation.

## Toxicology

Since the possibility of succinylcholine administration was of importance in this case, some toxicological aspects shall be addressed.

Succinylcholine (SUX) is a depolarizing neuromuscular blocking agent with rapid onset and short duration of action due to rapid hydrolysis into succinylmonocholine (SMC) by the plasma butyrylcholinesterase and subsequent slower degradation to choline and succinic acid (Figure 18.6). Homicides committed by injection of SUX may be difficult to detect due to the rapid metabolization of the parent drug.

A plasma concentration–time curve of SUX in a patient after intravenous administration of 2 mg/kg shows a triphasic elimination with apparent half-lives of 0.4, 1.2 and 8 minutes, and produces plasma levels of approximately 40, 2.4 and 0.5 mg/l at 0.5, 4.5 and 15 minutes respectively

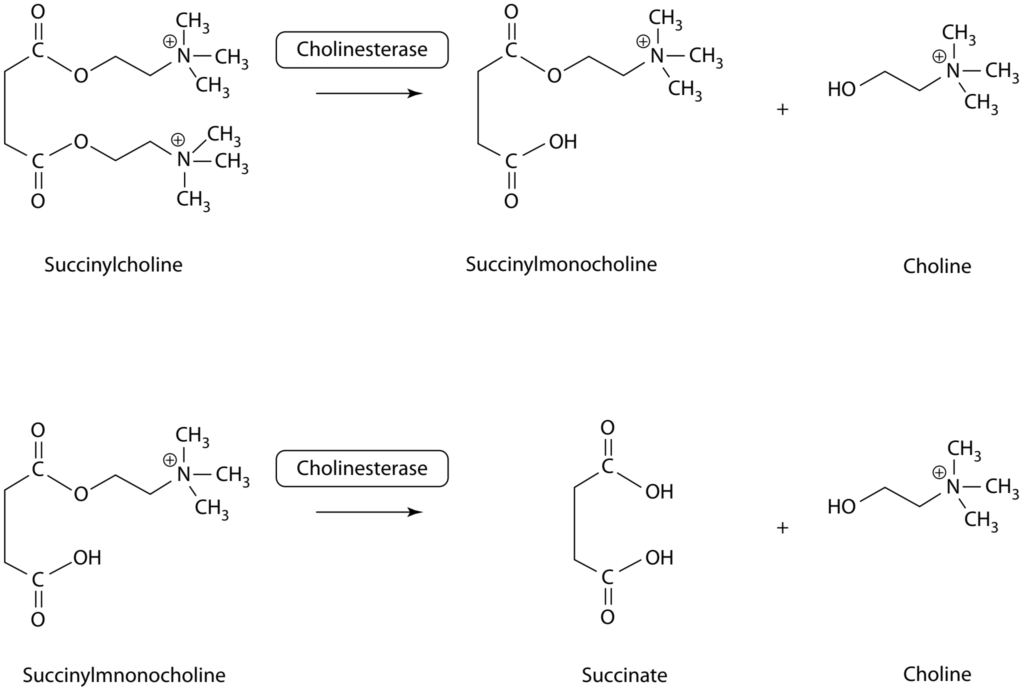

**Figure 18.6** Biotransformation of succinylcholine (SUX).

[98]. Plasma half-lives have been described as being in the range of seconds [78,162] – for example, after doses of 1 and 2 mg/kg, the respective half-lives were measured to be 25.4 and 26.3 seconds [83]. Analysis is further complicated by the fact that the compound will also undergo rapid *in vitro* hydrolysis in aqueous alkaline or even in acid solution at elevated temperature [65] and is absorbed to glassware during storage and sample preparation [33,131,165]. Several cases of SUX administration have been described, in suicides as well as in homicides [106,111,143,158].

Because of the rapid metabolization of SUX it has been suggested that succinic acid or SMC may be more reasonable analytes to indicate SUX administration in forensic cases. Succinic acid, both a metabolite of SUX and an endogenous dicarboxylic acid, has, however, been demonstrated not to be a suitable indicator of SUX exposure in forensic blood samples [116]. Additionally, in our own studies we found endogenous concentrations of SMC up to 300 ng/ml in negative control blood samples [33], a finding also described by others [100] and which therefore disqualifies SMC as a definite marker for SUX administration.

Ballard et al. [12] have described a rather complex analytical strategy for quaternary ammonium neuromuscular blocking agents including SUX and SMC using LC/MS, but without any validation data or results from authentic cases (only four SMC positive samples were described). Due to its analytically challenging character, determination of SUX content is not part of routine toxicological analyses. For further clinical studies, as well as applications in the forensic sciences, we developed a simple solid-phase extraction procedure for the simultaneous extraction of SUX and SMC with deuterated analogues as internal standards [91]. Paraoxon was found to be a useful stabilizing agent to prevent further enzymatic *in vitro* degradation. Degradation due to bacterial activity was not observed and is not described in the literature.

During the trial, two forensic toxicologists called by the court pointed out that the negative results of the toxicological analysis did not provide obvious evidence of administration of SUX. On the other hand, such an administration could not be excluded due to the quick degradation of the parent drug as described above. A third toxicologist excluded administration of the drug based on the negative results also for SMC. Although the presence of SMC no longer proves administration of SUX [33,100], it is absolutely not permissible to rule out such administration because of the absence of this metabolite.

## Conclusions

Although the cause of death could not be determined in the present case, several points raised suspicion and without the faked ECG it would probably have been difficult for the court to prove the husband guilty and sentence him. The faked ECG was an essential hint of 'foul play'. For the court, it was furthermore important to know that a lethal fall downstairs could be ruled out and that there was no clear anatomical cause of death, especially no pre-existing diseases. Therefore, autopsy findings that are not compatible with a typical traumatomechanism (negative autopsy findings) may also be essential to solve a case. A thorough evaluation of autopsy material in cases clearly based on a traumatomechanism is still essential as a basis for expert evidence.

It should further be mentioned that, without the first anaesthesiologist who was removed from the trial for reasons of prejudice, the case would probably have taken another course.

Of course, many questions in the case remain unanswered, which is common in homicide cases. If the husband had given details of what happened, the risk of being convicted for murder with a long-life imprisonment instead of for manslaughter would have been high.

## Educational messages

1. In each case it has to be proved whether the autopsy findings are compatible with the case history and findings at the scene; negative autopsy findings may be important to rule out a traumatomechanism that may be suggested at the first impression by scene findings.
2. Complete autopsy with detailed further investigations and multidisciplinary investigations are necessary in suspected crime affairs.
3. It is possible to fake an ECG with accurate date and time annotations. However, ventricular fibrillation is so individual that incidental conformity can be excluded.
4. Even considering negative toxicological findings, possible administration of SUX cannot be excluded due to the fast degradation of the parent compound. SUX metabolites (or SMC) are not definite markers for SUX administration.

# 18.2 Nurse-induced respiratory depression by succinylcholine

### *Frank Musshoff and Burkhard Madea*

The case discussed in Section 'Fall downstairs: Accident, homicide or natural death?' caused us to do extensive research about degradation and elimination of succinylcholine (SUX) and succinylmonocholine (SMC) and to develop a fully validated HPLC–MS/MS method for the simultaneous determination of SUX and SMC [91–97]. SUX is a bis-quaternary ammonium neuromuscular blocking agent routinely used as a muscle relaxant during surgery [12]. SUX is structurally as well as functionally related to the endogenous neurotransmitter acetylcholine (ACh), both activating postsynaptic ACh-receptors. While SUX is rapidly released from these receptors, its degradation by the specific acetylcholinesterase (EC 3.1.1.7) proceeds more slowly than that of ACh, thus causing a prolonged depolarization and desensitization of the postsynaptic membrane. Prior to expected paralysis, application of SUX leads to brief muscle convulsions (fasciculation). The effects start 30–60 seconds after i.v. application and continue for 3–10 minutes. As the whole skeletal musculature and also the respiratory muscles are paralyzed, spontaneous respiration is interrupted, so that immediate intubation and artificial respiration become necessary. If such measures are not taken, there is a risk of hypoxia resulting in circulatory arrest, brain damage and, finally, death. Therapeutically, SUX is used for the initiation of intubation; however, a hypnotic is given to the patients to avoid an extremely unpleasant paralysis when being awake prior to loss of consciousness. Additionally, typical side effects are increased salivation and boosted bronchial secretion.

SUX is degraded by the unspecific plasma cholinesterase (butyrylesterase; EC 3.1.1.8) to SMC. Finally, after complete hydrolysis of the ester bonds, one molecule of succinic acid and two molecules of choline are formed (Figure 18.7). The elimination half-life of SUX has been reported as 41 seconds [20] and 47.4 seconds [60]. These values were correlated with a window of detection of approximately 7 minutes following application of a single therapeutic SUX dose of 1 mg/kg [20]. The fast degradation of the parent drug within a few minutes after administration and its metabolization into endogenous substances lead to the reputation of SUX being an undetectable and thus perfect poison. Table 18.1 summarizes a selection of published fatal cases with suspicion of SUX application. Previously, an increase in choline or succinic acid concentrations as an indication for SUX application was discussed, but it cannot be regarded as a definite proof. Meanwhile, it is accepted that neither succinic acid nor choline is a suitable indirect indicator of SUX exposure in forensic cases [81].

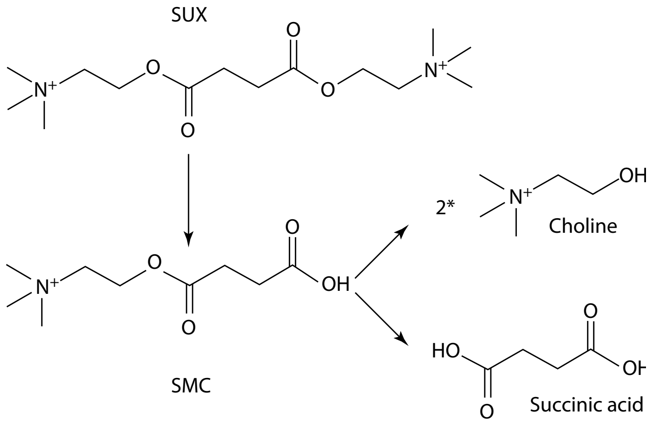

**Figure 18.7** Metabolism/degradation of SUX.

## Case report

A 78-year-old man was admitted to hospital with cardiac arrhythmias and pericardial effusions. He was transferred to an intensive care unit where a nurse was alone with him in the room. At 12.45 p.m., after talking to the nurse, he felt suddenly unable to move and breathe and started having gentle muscle convulsions and increased salivation. He was able to understand everything but could not speak, which filled him with fear of death. After a while, he regained the ability to breathe.

According to the nurse, the patient had had sudden seizures; she called for help and started giving ventilatory assistance using an Ambu™ bag. The first assistant doctor to arrive had the impression that the patient would have died without artificial respiration and was astonished by his regular circulation and sinus rhythm. When the senior physician arrived, the patient was already able to breathe normally and no intubation was necessary. The physician had no explanation for the respiratory arrest. However, he remembered more unexplainable respiratory

**Table 18.1** **Selection of published fatal cases with suspicion of an application of SUX**

| Case | Analytical results | Other findings | Author [ref] |
|---|---|---|---|
| Accidental injection to a 7-year-old boy | SUX detected in the area of injection<br>No unchanged drug in blood liver or kidney | – | Kato et al. [83] |
| Homicide of an anaesthetist's wife | Inconspicuous | High plasma choline concentrations | Küpper et al. [97] |
| Suicide of an anaesthetist | SUX 14.3 mg/ml in blood | Barbituric acid 106 mg/ml | Kuepper et al. [91] |
| Serial homicide; five victims were found buried in a rural area | SUX could not be detected in the victims | SUX identified in a syringe found near the corpses | Kuepper et al. [92] |
| Homicide of an anaesthetist's wife | Inconspicuous | Empty syringe of SUX found | Kuepper et al. [96] |
| Suicide of an anaesthetist | SUX 8.1 mg/L and SMC 3.7 mg/L in urine<br>Negative results in blood | Empty syringe of SUX found | Kuepper et al. [95] |
| Fatal cardiac arrest after anaphylactic reaction | None | Increased blood tryptase and immunoglobulin E values | Kuepper et al. [94] |

arrests happening in the department in the recent past, and it was always the same nurse who was caring for the patients or was in attendance. As the patient had had a respiratory arrest while conscious, he suspected that SUX, which was easily available in the hospital, might have been administered to the patient. After a phone call to us, he retained blood and urine samples for further toxicological analyses at about 5.50 p.m.; later it could not be clarified whether the urine was freshly collected or represented a pool sample from a urine bag.

## Results

The analyses of one plasma sample and three urine samples (three fillings of one sample?) revealed the results summarized in Table 18.2. Additionally, caffeine and bisoprolol (known medication) were detected in urine by a routine screening procedure via GC/MS. In Figure 18.8, authentic chromatograms are shown for the measurement of SUX and SMC. In the serum sample, we observed signal interferences for SMC and the calculated concentration has to be taken with caution. SUX and SMC concentrations in the second and third urine sample were a little lower compared to the first sample that had been analyzed after arrival. The other samples were submitted to one freeze/thaw cycle and were analyzed 12 days later.

**Table 18.2 Analytical results**

| Sample | SUX (µg/ml) | SMC (µg/ml) |
|---|---|---|
| Serum | n.p. | (0.05) |
| Urine no. 1 | 2.5 | 29.1 |
| Urine no. 2 | 2.0 | 21.5 |
| Urine no. 3 | 2.0 | 22.9 |

## Discussion

In the present case, chemical–toxicological results gave a definite proof for an administration of SUX responsible for the symptoms observed. In an additive-free blood/plasma sample taken approximately 5 hours post-injection, no SUX and only traces of SMC were detected. The sampling of urine was not well documented and it was not clear whether we received a fresh urine sample taken 5 hours and 5 minutes hours post-injection or a pooled sample accumulated approximately after 3 hours and 25 minutes and 5 hours and 5 minutes hours after application. However, in this urine – also collected in tubes without any additives – in addition to low concentrations of SUX, SMC could be identified. Given these results, the samples were not tested for other muscle relaxants.

The results correspond to data previously described in the literature. It has been shown that, after a single intravenous dose of 80–100 mg, SUX itself is detectable in plasma samples for approximately 10 minutes [100]. Generally, peak plasma concentrations up to 1 mg/ml are observed. In this clinical study, the fact that the blood samples taken were filled into tubes containing an aqueous paraoxon solution (100 ng/ml) has to be considered. The useful esterase-inhibitor paraoxon was utilized for stabilization of the drug. In non-stabilized samples, SUX was not found when sampling was performed later than 4 minutes post-injection, and a significantly decreased detectability was observed. In stabilized plasma samples, SMC was also detectable over a 6-hour period after injection.

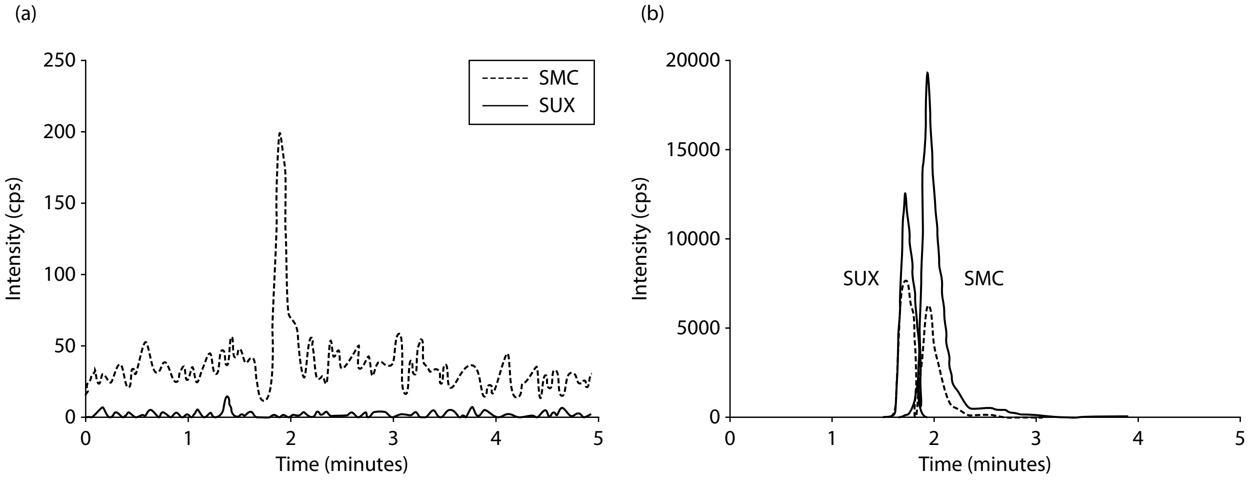

**Figure 18.8** Authentic chromatograms of the serum sample (a) and (b) a urine sample. For serum, the chromatogram only shows the quantifier transitions for SUX (solid line) and SMC (dotted line); the respective qualifiers did not rise above the background noise and were thus omitted for better visibility. For urine, each analyte is represented by both its quantifier (solid line) and its qualifier transition (dotted line). Internal standards are not depicted. In urine both analytes were detectable at concentrations of 2.0 mg/ml (SUX) and 21.5 mg/ml (SMC) (after recalibration; chromatograms were already published [95,10]).

The incidence of positive SMC findings in non-stabilized samples decreased to 50 per cent 4 hours post-injection, while after 6 hours, only one-third of tested samples still yielded a positive result.

Paraoxon stabilization of urine samples showed no effect, indicating that in this matrix esterase activity can be neglected. In fresh urine, SUX was detectable in 89 per cent of the samples 4 hours after injection; at 6 hours post-application a positive result was obtained in only 56 per cent of the samples. In comparison with the window of detection for plasma samples, it has to be assumed that the bladder was not completely emptied before in the weakened patients.

Recently, we have investigated the pharmacokinetic properties of SMC in surgical patients receiving 80–100 mg SUX via bolus injection [111]. SMC peak plasma concentrations were observed 0.03–2.0 minutes after application. In contrast to SUX, SMC was more slowly and more extensively distributed, featuring triphasic plasma concentration time profiles. With a terminal half-life of 1–3 hours, the window of detection for SMC in plasma was approximately 8–24 hours. However, this study used optimized sampling conditions (prompt sampling, instant stabilization, flash freezing), whereas less ideal conditions have to be expected in real forensic settings. Esterase activity on both SUX and SMC continues *in vitro* and especially postmortem and may lead to complete elimination of both analytes in blood and tissues. Especially in postmortem cases, more or less esterase-free urine seems to be the specimen of choice for the proof of an antemortem SUX application [106]. In tissue samples, caution must be taken regarding the possibility of false-positive SMC findings from interferences in the main ion transition [106,117,122,143]. Such interferences were excluded in the case presented.

In the present case, experts in toxicology and anaesthesiology came to the conclusion that, taking into account the chemical–toxicological findings and by exclusion of competing causes (medication, neurological, cardiac and infections of the respiratory tract), an administration of SUX had led to the observed symptomatology (i.e. gentle muscle convulsions; increased salivation; patient being unable to move and breathe while being conscious; regular blood circulation and sinus heart rhythm; individual being able to breathe normally again after a while). SUX was available on the hospital ward and the consumption quantity was not strictly monitored.

After this incident, the medical records of a further six patients who had earlier shown inexplicable apnoea led to the expert opinion of an anaesthesiologist being sought. In all cases the patients recovered following ventilator assistance and survived. According to expert opinion, SUX application was not excluded as the possible cause. It was noted that, in each case, the nurse concerned was dealing at the relevant times with the patients or was at least in their proximity and was also partially involved in their treatment. The woman enjoyed a high reputation with the doctors as well as with the nursing staff and took over with pleasure the care of seriously ill patients, particularly the care of artificially ventilated patients, to be in a position to apply her skills. Based on her training and long-standing activities, she knew about the impact of SUX and – in the opinion of the court – she accepted suffering and dangerous situations for patients deliberately to be able to take the necessary measures in one of her secretly caused critical situations.

She aimed to be the first one to benefit from the measures taken. According to the court, she wanted to present herself spectacularly as the first and decisive rescuer, to demonstrate her special skills and abilities, perhaps to gain a better job in the hospital. Because only proven facts could be taken as indications for the other cases, these charges were withdrawn, although in all of them culpability had also been considered at least possible. The nurse's sentence comprised compensation for personal suffering and imprisonment for 2 years on probation (§224(1) according to German law) because of dangerous physical harm, after a late admission of guilt. However, she revealed no details about dosages or anything else when admitting to the crime. The case appears similar to that of the fireman syndrome, in which a fireman committed arson to create a desperate situation which he himself could resolve, seeking recognition or heroism.

## 18.3 Homicidal poisoning with halothane

### ■ *Burkhard Madea and Frank Musshoff*

While several reports of criminal narcotization and even homicide as a result of chloroform poisoning have been published over the last 100 years [5,6,124,173], very few reports on halothane concentrations in tissue after i.v. or peroral (suicidal) or accidental ingestion have been published [22,120,136,139,157,160] and we were previously unaware of the double homicide by smothering with halothane-moistened towels reported here.

Halothane (2-bromo-2-chloro-1,1,1-trifluorethane) is a well-known volatile anaesthetic agent. However, considerable disadvantages are described by virtue of producing dose-related depression of the cardiovascular and respiratory system [76, 157].

### ■ Case report

According to the confession of one of the two suspected offenders, they facilitated a robbery from an antique dealer by poisoning of a 75-year-old man and his 82-year-old companion by forced inhalation of an anaesthetic substance. While one man diverted the antique dealer, the other moistened a towel with the agent from a dark green bottle containing 100 ml of the substance and then forcibly placed it over the mouth and nose of the woman. The woman became immediately unconscious and the free ends of the towels were knotted behind her head. Then the offender entered the room with the old man and, having prepared another anaesthetic-moistened towel, forcibly placed it over his mouth and nose and knotted the free ends of the towel behind his head in a similar manner (Figure 18.9a,b). Before the men left the house after the robbery, they removed the towels from the victims, both of whom died.

A few weeks later, a dark green 100 ml bottle still containing 25 ml of halothane was found, following information in the confession of one of the offenders. The offender thought that the contents of the bottle were ether.

## Autopsy findings

The autopsy (24 hours postmortem) of the 75-year-old man and the 82-year-old woman revealed signs of external violence with abrasion-type injuries on the nose, cheeks and perioral regions, some small haematomas and lacerations of the lips and oral mucosa, and petechial bleedings of the conjunctivae (Figure 18.10). Pre-existing diseases found were advanced atherosclerosis of the aorta and the great vessels and pulmonary emphysema in both victims.

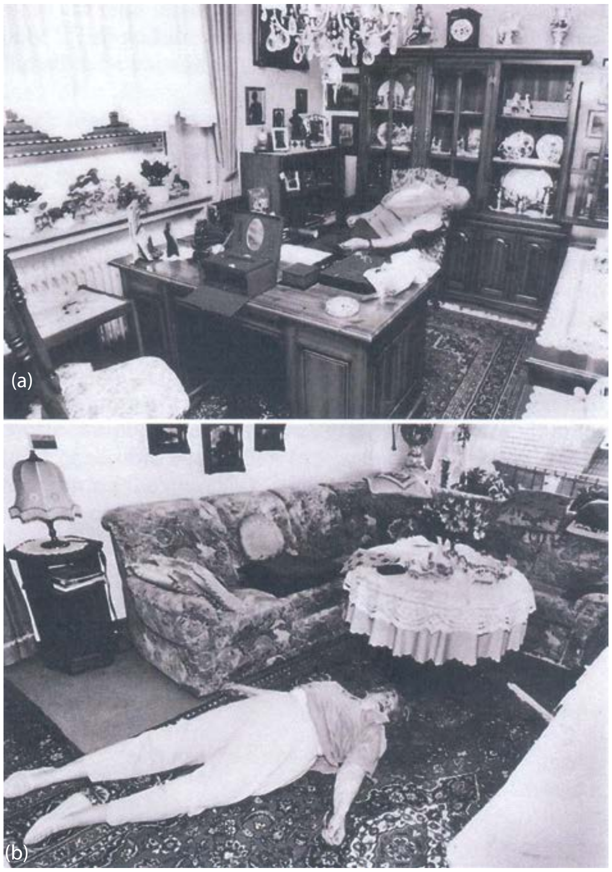

**Figure 18.9** (a),(b) Victims found at the scene.

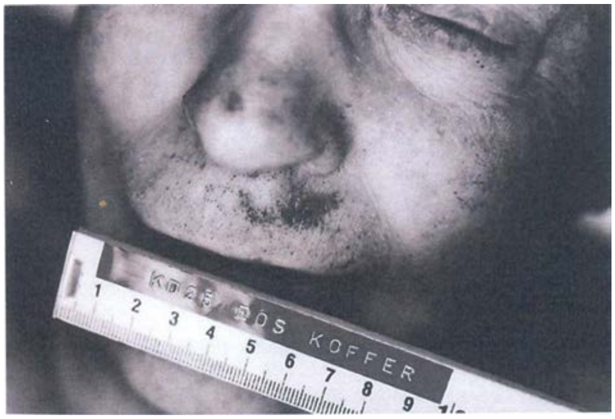

**Figure 18.10** Abrasion-type injuries of the nose and upper lip of the 75-year-old male.

## Toxicological findings

The headspace gas chromatograph used was a Perkin Elmer Model 8500 equipped with a flame ionization detector and a steel column (4 mm) packed with 5 per cent Carbowax 20M on a Carbopack B (60/80) mesh (Supelco/Inc.). The carrier gas was $N_2$ at a flow rate of 40 ml per minute. Temperature settings were 170°C for the injector, 250°C for the detector, and the oven temperature was programmed from 70°C to 170°C. Prior to injection of 1 ml or 1 g of homogenized biological sample, respectively, 2 g of sodium sulfate was added to a 20 ml headspace vial and incubated for 45 minutes at 80°C. Aqueous calibration standards of halothane were prepared in the same way and the calibration curves showed a good linearity over a concentration range of 1–100 mg/l with a coefficient of correlation of 0.996. Analysis of spiked blood samples revealed no matrix effects.

The analysis of halothane was performed 3 months postmortem and the specimens had been stored at −20°C until analysis.

The toxicological investigations on blood, other body fluids and tissues revealed the halothane concentrations given in Table 18.3. Although the obtained blood concentrations in the presented case were much lower than during halothane anaesthesia (22–84 mg/l), there is no doubt that both victims died because of forced inhalation of halothane. Halothane was rapidly distributed, mainly into the brain, but the inhalation was survived for only a few minutes. Both victims must have become rapidly unconscious (within seconds) and there were no defence injuries.

The minimal lethal dose of halothane after ingestion or inhalation is given as 10 ml. Blood concentrations in these cases were reported to be in the range 7–310 mg/l. In a case of suicidal poisoning with halothane, 35 ml were ingested orally [157] and much higher tissue concentrations were found (Table 18.3). In a case of i.v. injection of halothane survived for several hours, blood concentrations were

**Table 18.3 Halothane levels in blood and tissue in the victims compared to those found in the literature**

| | S 195/96 female 82 years | S 196/96 male 75 years | Spencer / Green [160] male 19 years | Berman/ Tattersall [22] male 42 years |
|---|---|---|---|---|
| Route of exposure | Forced inhalation of halothane | | Oral ingestion | i.v. injection |
| Survived period | A few minutes | | ? | Some hours |
| Blood (mg/l) | 6.7 | 3.4 | 650 | 7.9 |
| | 8.3 | 3.2 | | |
| Liver (mg/kg) | 1.7 | 21.3 | 880 | |
| Kidney (mg/kg) | 11.7 | 14.5 | | |
| Brain (mg/kg) | 103.6 | 120.2 | 1560 | 1.58 |
| Urine (mg/l) | 0.0 | 0.0 | 20 | |
| Bile (mg/l) | 8.6 | 1.6 | | |
| Stomach (mg/kg) | | 10.3 | 240 | |

slightly higher than in our cases [22]. In cases of chloroform poisoning, tissue concentrations were also much higher after oral ingestion than after forced inhalation [130].

In the case presented, the 100 ml bottle contained only 25 ml halothane and the missing volume of 75 ml could be assumed to be enough to induce the lethal outcome in two persons after forced inhalation. Halothane was inhaled in much higher concentrations than during anaesthesia (0.5–1.5 vol%) with a rapid increase of concentration. However, in our case additional lethal factors, namely suffocation by occlusion of the mouth and nose (abrasion-type injuries of the face) and lethal arrhythmia secondary to cardiac sensitization to catecholamines during the assault with forced inhalation of halothane, have to be taken into account [76]. Furthermore, both victims were much older than in other reported cases and also revealed accompanying pre-existing disease. Additionally, a slight decrease of concentration after tissue/fluid storage has to be taken into consideration, because the material was not stored under strict anaerobic conditions (no suspicion of volatile poisons during the autopsy).

The cause of death in both cases was given as halothane poisoning due to forced inhalation, and the manner of death was homicide. Both suspects were convicted of aggravated robbery with fatal outcome according to §§250, 251 of the German penal code and sentenced to long-term imprisonment (11 years 9 months and 14 years respectively).

## 18.4 Cyanide fatalities

### *Frank Musshoff and Burkhard Madea*

Deaths from cyanide poisoning are thought to be relatively rare, largely because the availability of cyanide is restricted. Nevertheless, cyanide is one of the most rapidly acting poisons known, and it accounts for both suicides and homicides [39,58,127].

Here, we report five cases which were observed within a few months.

### Case 1

A 38-year-old goldsmith was last seen alive entering his shop early on a Sunday morning. The next morning, he was found dead by his wife, lying face down on the floor of a very confined toilet room beside his workshop. His head was surrounded by a large pool of blood. The physician who issued the death certificate performed an external examination of the corpse, assumed a lethal injury due to a fall, and determined the manner of death to be accidental.

Additional police investigations after the performance of the autopsy revealed a history of psychiatric disorder and a recent announcement by the decedent's wife that she intended to get divorced. Although a thorough examination of the scene failed to yield a farewell letter, this information focused attention on a plastic vial labelled 'Farbvergoldungsbad', which had been found in the clothing of the deceased. Toxicological analysis of the contents disclosed pure sodium cyanide.

Autopsy was performed 3 days later. External examination revealed livor mortis of inconspicuous colouration distributed in keeping with the initial position of the corpse, petechiae and purpura in the area of livor mortis, intense congestion of the face, a discrete bruise at the front of the neck, and a linear abrasion on the right lateral aspect of the neck. Internal examination showed an underlying fracture of the right superior horn of the thyroid cartilage, but no further injuries of the soft tissues of the neck. Neither penetrating bleeding wounds of the skull nor defence wounds were found. The remainder of the autopsy revealed a fatty liver and pronounced visceral congestion, in particular involving the buccal, pharyngeal, laryngeal, oesophageal and gastric mucosae; no erosions, ulcerations or necrosis were observed. The stomach contained a few millilitres of a viscous brown fluid without suggestive additional compounds. A characteristic pungent odour, usually likened to that of bitter almonds, was not noted.

Toxicological analysis of the specimens obtained at autopsy yielded the following cyanide concentrations: blood 80.9 mg/l, gastric contents 1.26 g/l, lung 46.3 mg/kg, brain 4.8 mg/kg, kidney 0.89 mg/kg, liver 17.6 mg/kg, bile 21.3 mg/l. The results of assays for other drugs were negative. The blood alcohol concentration was 0.1 g/dl.

These findings provided sufficient evidence that death was attributable to cyanide intoxication. The origin of the injuries at the right side of the neck could not definitely be elucidated but was related to an agonal fall in the small toilet room. The distribution of cyanide in the specimens examined indicated oral ingestion, and the manner of death was finally determined to be suicide.

## Cases 2 and 3

A physician and his wife, both 71 years old, were found dead at home by their daughter. The body of the woman, clad in a nightdress, lay on her bed; the body of the man, dressed in pyjamas, lay on a mattress in the immediate vicinity. External examination of both corpses revealed no major injuries. Livor mortis of purple colouration was observed in the dependent areas of the bodies. A paper providing precise advice on how to handle the dangers related to cyanide was found on the cupboard, and the wastebasket contained two vials filled with cotton wool. A farewell letter signed by both decedents indicated as a motive that the woman was seriously handicapped after incomplete recovery from a stroke and that her husband feared being overburdened by the demands of the appropriate care. Autopsy was not carried out.

The two vials, which were supposed to have contained cyanide, and two blood samples were submitted for toxicological analysis. The examination of the vials yielded positive results for cyanide. Analysis of the blood samples quantified cyanide concentrations of 6.1 mg/l and 8.6 mg/l respectively, which indicated severe intoxication and provided adequate evidence to attribute both deaths to cyanide toxicity.

With regard to the circumstances elucidated by the police investigation, the manner of death was determined to be suicide.

## Case 4

According to the report of the police investigation, the 70-year-old wife of a chemist had experienced depression, had had serious financial problems in the family, and had several times announced the intent to commit suicide. From her husband's laboratory, she had secretly removed a white powdery substance, which had earlier been described by her husband as highly toxic, and had ingested a spoonful while her husband and a friend were present in the adjoining room. When she was found a few minutes later and admitted to having consumed the powder, her husband instantly suspected sodium cyanide. The woman experienced convulsions and, despite immediate attempts at resuscitation, died approximately 2 hours later.

Autopsy showed severe coronary atherosclerosis with significant luminal narrowing caused by fibrofatty plaques and two surgically implanted aortocoronary bypass vessels, but no complete occlusion and no recent myocardial infarction. The colouration of livor mortis was not suggestive. The gastric mucosa did not show a striking haemorrhagic appearance, erosions or ulcers. A strong bitter odour emanating from the viscera was not recognized. The cause and manner of death could not be determined unequivocally at gross examination.

Toxicological analysis of the blood demonstrated therapeutic concentrations of oxazepam. The assays for cyanide yielded the following levels: blood 42.5 mg/L, brain 2.54 mg/l, gastric contents 1.2 g/l. The white granular powder taken by the woman was proved to be sodium cyanide.

It was concluded from the toxicological findings that the deceased had access to cyanide, had ingested the substance orally, and had died of cyanide intoxication.

## Case 5

The deceased was a 29-year-old woman who worked as a laboratory technician in a metal-processing factory. After having a cappuccino with her colleagues, she experienced seizures and collapsed. When the emergency doctor arrived a few minutes later, she was already comatose. Because no organic disease that could have caused the seizures was diagnosed, the workplace was taken into consideration, and the suspicion of intoxication arose.

Four hours after the woman was admitted to hospital, specimens of blood and stomach contents were obtained for toxicological examination. The analysis revealed a blood cyanide level of 3 mg/l. Death occurred 4 days later.

The police investigation disclosed that a male colleague had fetched the cappuccino from an automated buffet in the factory. According to witnesses, the drink had had a pungent putrid odour resembling ammonia and a bitter disgusting taste. Because the deceased was reported to have drunk only a sip of the cappuccino, and furthermore had access to cyanide in her professional work, the manner of death was assumed to be suicide.

Several months later, the colleague who had passed the cappuccino to the deceased was suspected of having poisoned the woman by means of a lethal dose of cyanide. He was convicted of murder and sentenced to long-term imprisonment on the basis of circumstantial evidence, i.e. a specialist's report on the changes in quality and odour of a cappuccino caused by the addition of cyanide. The reason for the crime was embezzlement of gold in the factory by the suspect.

## Chemistry and epidemiology

Cyanide exists in many different forms, the most common being hydrogen cyanide (HCN) and cyanic salts (potassium cyanide, sodium cyanide, calcium cyanide), which can react with acid to release CN. Also, some aliphatic nitrile compounds (i.e. acrylonitrile, acetonitrile, propionitrile) and aliphatic thiocyanates can release cyanide by hepatic metabolism after absorption, resulting in the delayed onset of cyanide poisoning. Thus, cases have been reported in which patients experienced the symptoms of cyanide poisoning hours after the ingestion of acetonitrile-based artificial nail polish remover [35,118,171].

Cyanide salts are generally colourless solids; HCN (prussic acid) is a colourless gas at room temperature. Cyanide compounds have a faint, bitter almond odour, detectable at a threshold of 0.2–5.0 ppm. The ability to smell cyanide is a genetically determined trait that is absent in up to 50 per cent of the population. Cyanide may be released from various compounds by chemical reactions or by pyrolysis. Significant blood cyanide levels have been found in many fatal and non-fatal cases of fire-related smoke inhalation [17,104], and the risk of cyanide intoxication due to smoke inhalation injury is probably underestimated. The inhalation of HCN can lead to weakness and loss of muscle coordination, impairing the escape from a fire.

As for other sources of cyanide, cigarette smokers have been found to have mean whole blood cyanide levels of approximately 0.41 mg/l, more than 2.5 times the mean in non-smokers [13]. Natural sources of cyanide include amygdalin and similar cyanogenic substances found in a wide variety of plants [70,126].

## Toxicity, pathophysiology and clinical presentation

Cyanide toxicity has been extensively reviewed [2,32,70,144] and is therefore summarized only briefly here. Cyanide is absorbed through the lungs, gastrointestinal tract and skin. Symptoms can occur within seconds of HCN inhalation and within minutes of the ingestion of cyanide salts. The onset of symptoms may be delayed up to 12 hours after the ingestion of cyanogenic glycosides, nitriles or thiocyanates. The absorption time depends on the pH and solubility of the cyanide-containing compound. After absorption, cyanide is rapidly distributed by the blood circulation throughout the body.

Cyanide exerts its toxic effects by combining with the ferric iron in cytochrome oxidase, which inhibits the utilization of cellular oxygen (Figure 18.11). The blockade of the cytochrome oxidase system causes anaerobic metabolism, which results in lactate production and severe metabolic acidosis. Cyanide also inhibits other enzymes and can combine with certain metabolic intermediates. Eighty per cent of absorbed cyanide is detoxified in the liver by the mitochondrial enzyme rhodanese, which catalyzes the transfer of sulfur from a sulfate donor to cyanide, forming less toxic thiocyanate, which is rapidly excreted in urine. Other detoxification pathways include reaction with hydroxycobalamin (vitamin B12b) to form cyanocobalamin (vitamin B12). Only a small amount of cyanide is eliminated as carbon dioxide by expiration, along with small amounts of HCN.

Acute cyanide poisoning results primarily in central nervous system effects with early stimulation, which is quickly followed by depression. Early symptoms include lightheadedness, giddiness, tachypnoea, nausea, vomiting, suffocation, confusion, restlessness and anxiety. Stimulation of the peripheral chemoreceptors produces increased respiration. Otherwise, stimulation of the carotid body receptors slows the heart rate. These changes may be followed by hypoventilation, progressing to apnoea and myocardial depression. The result is hypotension and shock, which may rapidly be fatal. The predominance of anaerobic metabolism induces a decrease in the adenosine triphosphate/adenosine diphosphate ratio and alters energy-dependent processes such as calcium homeostasis.

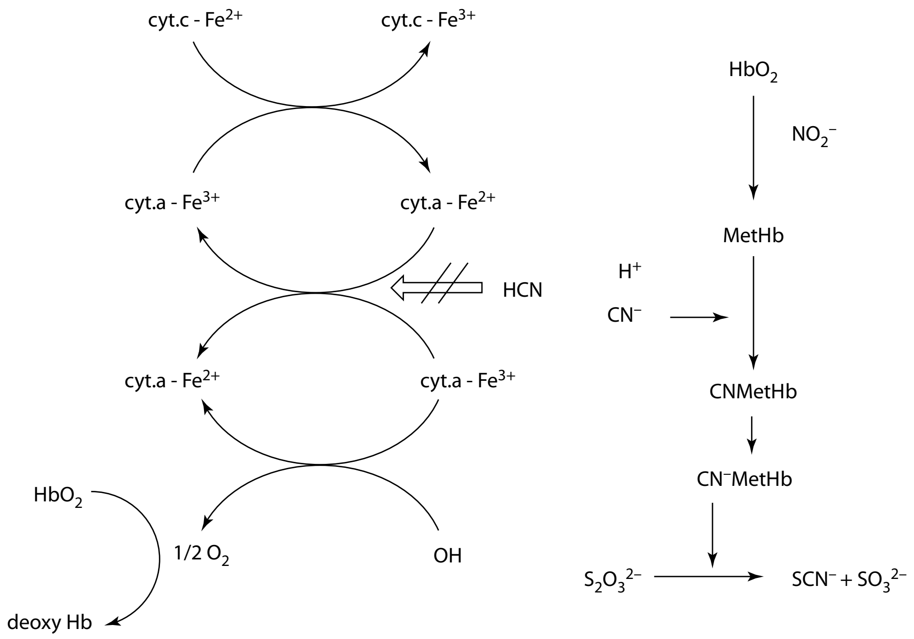

**Figure 18.11** Mechanism of cyanide poisoning and the action of antidotes.

Disruption of calcium regulation with resultant changes in neurotransmitter releases can alter the electrical activity in the brain and may be responsible for neurotoxic effects such as tremor and convulsion. In summary, the signs and symptoms of acute cyanide toxicity reflect cellular hypoxia and may initially be non-specific, generalized and non-diagnostic. Cyanide should be included in the differential diagnosis of rapid onset of coma and, in particular, when coma and metabolic acidosis appear together.

The treatment of cyanide poisoning begins with removing the patient from exposure, administering 100 per cent oxygen, and giving aggressive cardiorespiratory support and an antidote. Inhalation of amyl nitrite has been recommended as a first-aid measure, followed by the intravenous administration of sodium nitrite and/or sodium thiosulfate. Nitrites induce methaemoglobinaemia, which detoxifies cyanide by forming cyanomethaemoglobin. Thiosulfate serves as a sulfur donor in the rhodanese-catalyzed conversion of cyanide to less toxic thiocyanate. Methaemoglobin levels should be monitored serially during treatment. Blood cyanide levels are useful in confirming the diagnosis of intoxication, but therapeutic interventions must usually be initiated before the results of analysis are available.

Especially in forensic cases, an accurate assessment of the duration of the ability to act after lethal cyanide poisoning can be of crucial importance in reconstructing the cause of poisoning. Vock et al. [174] investigated 27 cases of lethal oral intoxication with cyanide with regard to the post-intoxication ability to act. In most cases, the capacity lasted only a few seconds to 1–2 minutes. However, in some cases, the capacity to act lasted for 5–10 minutes. Subjects with cyanide intoxication may be able to give a report about the incident, remove a poison container, or walk for a few hundred metres.

## Toxicological findings

Whole blood cyanide levels are higher than plasma levels because of the concentration of cyanide in the red blood cells. Correlation of symptoms to whole blood levels is misleading because the effect of cyanide depends on the intracellular concentration at the cytochrome oxidase binding sites and the duration of poisoning. Peak whole blood cyanide concentrations lower than 0.2 mg/l usually do not cause symptoms, although poisoning has sometimes occurred at lower levels [2]. Whole blood cyanide levels in smokers may reach 0.4 mg/l without causing symptoms. At cyanide concentrations between 0.5 and 1.0 mg/l, untreated patients may be conscious, flushed and tachycardic. Stupor and agitation can appear with peak blood levels between 1.0 and 2.5 mg/l. Cyanide levels greater than 2.5 mg/l are associated with coma and are potentially fatal without treatment. According to Baselt and Cravey [18], the minimal lethal dose has been estimated to be 100 mg for HCN, 150 mg for sodium cyanide and 200 mg for potassium cyanide. Nevertheless, factors such as age, body mass, state of health, and mode of ingestion may influence these values, and survival after the ingestion of larger quantities has been reported. In this respect, 37 mg of HCN has been fatal, whereas recovery has been reported after the ingestion of 300 mg [7]. The blood cyanide concentrations in 32 fatal cases ranged between 0.4 and 230 mg/l (mean 37 mg/l) [137]. In comparison with these published data, the blood cyanide levels in our cases (3.0–80.9 mg/l) provided sufficient evidence to determine that death was due to cyanide toxicity.

Important for the interpretation of toxicological findings is the fact that cyanide levels may drop as a result of degradation of cyanides into less toxic components if the postmortem interval prior to autopsy is too long [15,16]. Thus, in fatalities suspected to result from cyanide poisoning, the autopsy and toxicological analysis should be performed as soon as possible after death. Postmortem endogenous cyanide formation in tissue is less important, because the amounts formed are not significant. Postmortem cyanide peak concentrations of only 0.2 mg/L are not likely to be confused with fatal cyanide poisoning [14]. However, because of the instability of cyanide at a storage temperature of 4°C, it has been recommended that blood samples should be analyzed immediately after collection; otherwise, they should be stored directly in a freezer (–18°C) until analysis [163].

## Indicative circumstances of cyanide poisoning

Most cases of occupational cyanide poisoning are easily recognized because of the known circumstances. In professional persons having access to cyanide, the history often indicates the correct direction of the investigation. Recent reviews concerning cyanide-related deaths have suggested that the suicidal use of substances is usually limited to specific occupational groups (Table 18.4) [26,67,149,175]. In Case 1 here, however, the circumstances of the scene and in particular the initial death certificate had focused attention on the evidence that suggested an accidental fall, so that investigation of the history and examination of the locus with regard to intoxication was neglected. Nevertheless, the autopsy findings and laboratory tests in association with the evidence discovered subsequently were in keeping with one another and left no reasonable doubt that the manner of death was suicide.

In agreement with the literature, the individuals involved in these five fatalities, without exception, had cyanide at their disposal. The goldsmith in Case 1 and the accused in the homicide Case 5 had professional access, as evidenced by the special ingredients found at their workplace. The double suicide using so-called suicide pills was committed by a physician and his wife. In Case 4, the access was related to the profession of the decedent's husband.

**Table 18.4 Occupational access to cyanide**

| |
|---|
| Acrylate or nitrile manufacturers and users |
| Blacksmiths |
| Dyeing |
| Electroplating |
| Metal heat treatment processes (case hardening) |
| Mineral refining and extraction |
| Paper and textile manufacturers |
| Pest control |
| Photography |
| Plastic manufacturers and users |
| Printing (textile) |
| Tanning |
| Working in laboratories |

### Pathological findings

The autopsy findings in cyanide poisoning are, in general, non-specific [58]. Unlike the characteristic and easily detectable pink hypostasis of carbon monoxide poisoning, the bright pink or lilac colouration ascribed to cyanide poisoning is difficult or impossible to detect at autopsy. Furthermore, the additional presence of a pronounced cyanosis may further obscure the discolouration [172]. In addition, a striking haemorrhagic appearance of the gastric mucosa has been described, which may be associated with grossly oedematous gastric rugae, producing a velvety appearance of the gastric lining. The underlying histological changes constituted vacuolation of the basal mucosal cells, basal cell dissolution, desquamation of surface epithelial cells, and discrete inflammatory infiltration of the submucosa.

In keeping with the literature, the cases reported here emphasize that the diagnosis of cyanide intoxication cannot reliably be established at autopsy because the indicative morphological findings are difficult to recognize unequivocally or may even be missing.

Injuries ex post attributable to a fall during agony, as in Case 1, have been reported in approximately 15 per cent of cases [129].

At autopsy, considerable caution should be exercised in an attempt to smell cyanide, because this procedure is associated with the risk of inhaling cyanide fumes [4].

## 18.5 Homicide in the bathtub

### *Peter Schmidt and Burkhard Madea*

Homicide in the bathtub and homicide with subsequent submersion of the corpse are rare events. In a retrospective survey of 36 fatalities in the bathtub in Ghent from 1934 to 1983, Devos et al. [42] reported two homicides. In a series of 42 deaths in the bath in Copenhagen and Gothenburg from 1961 to 1969, Geertinger and Voigt [63] encountered one homicide. Retrospective analysis of 245 fatalities in the bathtub in Hamburg from 1971 to 1988 revealed 13 homicides (5.3%) [169]. Several cases of homicidal electrocution in the bathtub have been reported in detail [134,153,179]. There is, however, a lack of more comprehensive analysis covering the variety in the range from homicides actually occurring in the bathtub to mere deposition of the corpse to cover traces. Trübner and Püschel [169] claimed that the pattern of injury was indicative of the manner of death. In a case of manual strangulation with subsequent submersion of the corpse in a bathtub described by Spitz [161], however, initial examination of the body at the scene had only revealed two small whitish areas on the side of the neck while autopsy of the dried body showed unequivocal nail marks.

Our own retrospective analysis of the fatalities investigated in the Institute of Forensic Medicine, University of Cologne, from 1980 to 1993 revealed 215 fatalities in the bath, i.e. fatalities which had occurred in the bathtub or corpses found there. These fatalities comprised 11 homicides (5%). A survey of our material is given in Table 18.5. Ten victims were female, one was male. The ages of the deceased ranged from 13 to 63 years, and the age group 20–40 years accounted for most of the fatalities.

In six of these cases, the bathtub was filled with water and empty in four when the corpse was encountered. In one case the circumstances indicated that the bathtub had originally been filled with water that had been let out before the discovery of the corpse. Although the mouth and nose were found without exception to be beneath the surface of the water, the autopsy revealed signs of drowning in only four fatalities (one of them coming out of an empty bathtub). The diagnosis of lethal drowning was made in two cases based on the circumstances of death, complete autopsy and negative toxicological screening.

In five cases, death was attributed to strangulation and in four cases to sharp violence. In eight cases, findings of severe miscellaneous violence were encountered at the autopsy, leaving no doubt as to the manner of death. In contrast, two fatalities showing only discrete signs of injury on external examination were not recognized as homicides at the scene. Four corpses had merely been deposited in the bathtub, and waterlogging had not actually influenced the lethal outcome. Four homicides were committed partly or completely in the bathtub (drowning, strangulation, ligature strangulation with the tube of the shower). One victim was submerged in the bathtub to ensure death after stabbing. In two cases the scenario raised the question of whether the victim had been put into the bathtub after manual strangulation to ensure death by electrocution.

At the time of death, three of the victims were under the influence of alcohol, with the BAC in the range 0.17–2.73 g/l and indicating severe impairment only in one case. Toxicological screening without exception revealed negative results.

**Table 18.5 Homicides in the bathtub investigated during the period 1980–1983 (Institute of Forensic Medicine, University of Cologne)**

| Age | Sex[a] | Water in tub[b] | Head submerged[c] | Signs of drowning[d] | Violence | Cause of death | BAC[e] | Tox[e] |
|---|---|---|---|---|---|---|---|---|
| 63 | F | + | ↓ | – | Strangulation | Strangulation | Neg. | * |
| 29 | F | + | ↓ | + | Repeated blunt force | Drowning + bone marrow and fat embolism | Neg. | Neg. |
| 22 | M | + | ↓ | – | Abrasions, contusions and lacerations of the skull, 98 stab wounds | Stabbing | Neg. | Neg. |
| 37 | F | + | ↓ | + | Haemorrhages in the soft tissue of the back, strangulation | Strangulation | 2.73 | Neg. |
| 13 | F | – |  | – | 17 stab wounds of the chest and abdomen, binding and gagging | Stabbing | * | * |
| 32 | F | – |  | + | Contusion of the skull, haemorrhages in the soft tissues of the back and the arms | Drowning | Neg. | Neg. |
| 39 | F | – |  | – | 5 stab wounds of the chest, injury of the head due to a slaughtering gun, contusions of the skull | Stabbing + shooting | Neg. | * |
| 26 | F | let out |  | – | Strangulation, contusion of the skull, incised wound of the flexor surface of the wrist | Strangulation | Neg. | * |
| 32 | F | + | ↓ | – | Strangulation, incised wound of the neck | Strangulation | 1.27 | Neg. |
| 55 | F | + | ↓ | + | Manual strangulation, contusion of the skull | Manual strangulation | Neg. | * |
| 41 | F | – |  | – | 9 stab wounds of the neck and the trunk | Stabbing | 0.17 | Neg. |

*Source:* From Schmidt P and Madea B. *Forensic Sci Int* 1995;**72**:135–146. [148]

*Abbreviation:* BAC = blood alcohol concentration.

[a] F = female, M = male

[b] + = water in tub, – = tub empty

[c] ↓ = head submerged in water

[d] – = absent, + = present

[e] * = not investigated

To illustrate the great variability of scenes encountered, three cases are reported briefly here.

## Case 1

In this case, a 32-year-old woman was found dead by her husband in the bathtub, lying undressed, face down, with her mouth and nose beneath the surface of the water. The electrical cord of a vibrator was wrapped around her neck. On the floor of the living room a pool of blood was encountered with a trace of dragging leading in the direction of the bathroom. The autopsy revealed a ligature mark of the neck, corresponding paleness of the mucous membrane of the gullet and multiple petechiae of the conjunctivae. In addition, a deep incised wound of the throat was found, but there were no signs of blood aspiration, air embolism or marked anaemia of the internal organs due to haemorrhage. Findings indicating drowning or defensive injuries were not encountered.

In court the perpetrator confessed that, in the course of a fight with a sexual background, he had first strangled the woman with the electric cord, inflicted the incised wound of the neck and then submerged the corpse in the bathtub.

## Case 2

In the second case, a 37-year-old woman was found dead in her bathtub with her mouth and nose submerged in water and abundant white foam escaping from the mouth. The hand shower tube was wrapped around her face and neck (Figure 18.12). The autopsy showed a pale ligature mark exactly corresponding to the position of the tube (Figure 18.13), haemorrhages of the skin and the muscles of the neck, fractures of the inferior horns of the thyroid cartilage and the cricoid cartilage, severe haemorrhages of the mucous membrane of the larynx and the anterior surface of the cervical spinal column, and petechiae of the conjunctivae. In addition, haemorrhages of the skin were found in the sacral region and over both elbows as well as haemorrhages of the soft tissues covering the thoracic and lumbar spinal column and the left clavicula. The lungs were large and bulky and on cut sections had a brick-red appearance with large quantities of foamy oedema fluid flowing from the cut surfaces.

The content of the stomach was intermingled with water. Toxicological analysis revealed a BAC of 2.73 g/l but was negative for drugs.

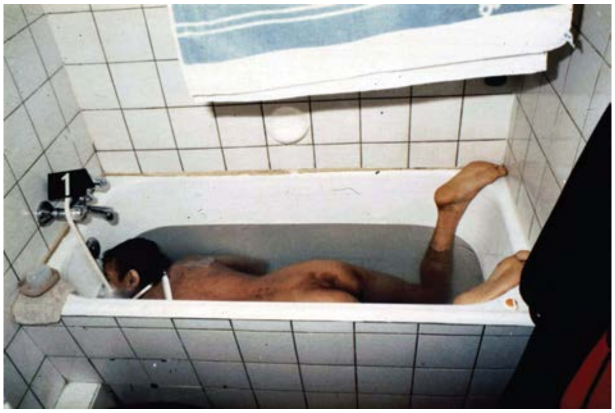

**Figure 18.12** Case 2 showing the shower tube wrapped around the face and neck. White foam has escaped from the mouth.

## Case 3

In the third case, a 55-year-old woman was found by her son face down submerged in her bathtub, with her mouth and nose beneath the water. He let out the water and sat his mother up before calling the police. Faint haemorrhages and scratches of the skin and the neck recognized on external examination at the scene were supposed to be related to a necklace. The bathroom showed no signs indicating that a struggle had taken place. The autopsy revealed multiple haemorrhages in the muscles of the neck, fractures of the superior horns of the thyroid cartilage surrounded by haemorrhage, haemorrhages of the mucous membrane of the larynx, numerous petechiae of the skin of the face, the conjunctivae and the pleural surfaces of the lungs, and in addition signs of agonal swallowing of water. Defensive injuries were not encountered.

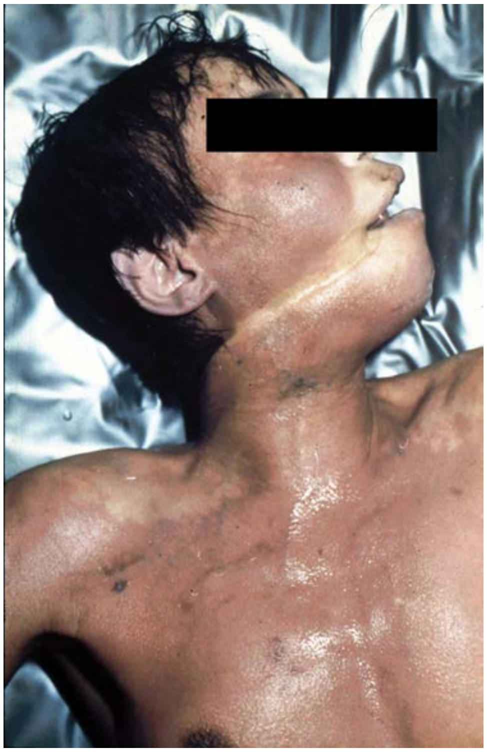

**Figure 18.13** Pale ligature mark of the face exactly corresponding to the position of the tube. Case 2, see Figure 18.12.

## Autopsy analysis

As stated earlier, homicides committed in the bathtub or mere deposition of the victim of a homicide in the bath are rare events. In the forensic literature (Table 18.5 [23, 56,86,134,150,151,153,161,179] and Table 18.6 [31,42,52,63, 66,82,89,90,99,135,152,169]) attention has been focused on the problems of evidence in cases of homicidal drowning or electrocution. The analysis of our autopsy material, however, is intended to give a survey of the entire spectrum encountered and to elucidate the problems in establishing the diagnosis of cause and manner of death apart from drowning and electrocution.

In our material young women aged 20–40 years make up most of the victims of a homicide found in the bathtub. This is in good agreement with the data from the literature summarized above (sex: 21 females, 4 males; age: 4 children, 11 adults aged 20–40 years, 6 adults >40 years).

At autopsy, Trübner and Püschel [169] found signs of drowning in 40.8 per cent, Devos et al. [42] in 57 per cent and Geertinger and Voigt in 74 per cent [63]. In our material comprising only homicides, autopsy revealed evidence of aspirating, respectively swallowing water in 36 per cent of the cases. These autopsy findings provided important information. In every case they indicated that the victim was still alive when entering or being put into the bathtub. In one case the autopsy findings of drowning brought about the suspicion of homicide, and in two cases drowning was the cause of death. Stimulated by the case of GJ Smith (see Table 18.5), the difficulties of proving homicidal drowning in the bathtub have already been discussed in detail [42,56,86,121,151]. The remaining causes of death comprise strangulation and sharp violence. In comparison, analysis of the literature cited in the tables above renders the following distribution: 14 by strangulation, 10 by drowning, 6 by electrocution, 5 by blunt force and 5 by sharp force. The lack of homicidal electrocution in our material is due to the fact that this cause of death was not proved beyond doubt in any case. The problems of verifying lethal electrocution and of distinguishing between suicide, accident and homicide under the conditions of waterlogging are well known [29–31,36,52,66,82,115,134,135,141,152–154,179,181].

Although findings of severe miscellaneous violence allowed the unequivocal diagnosis of homicide in eight cases, one case of drowning and one case of manual strangulation were not recognized before the autopsy (possible reasons are discussed below). In the literature, nine additional cases [23,56,86,99,161,169,179] are reported in which the accurate diagnosis of homicide could not be established at the scene (four by drowning, three by

**Table 18.6 Homicide in the bathtub – Case reports**

| Author [ref] | Cause of death | Case report |
|---|---|---|
| Betsch and Giesel [23] | Manual strangulation | Deposition of the victim in the shower tray, then suicidal electrocution of the perpetrator; at first assumption of two homicides |
| Engelhardt [56] | Drowning (3 times) | GJ Smith (serial killer), drowning by grasping the feet and pulling underwater by them |
| Kosa and Viragos-Kis [86] | Drowning | At first misjudged as CO intoxication |
| Püschel [134] | Electrocution | Detailed police investigation and confessions of the perpetrators blaming one another |
| Schneider [150] | Manual strangulation (+ electrocution) | Corpse put into the shower tray for dismembering |
| Schneider [151] | Blunt force + drowning<br>Electrocution + drowning | |
| Schwerd and Lautenbach [153] | Electrocution | Characteristically shaped electrical burns caused by a self-built conductor |
| Spitz [161] | Manual strangulation | Recognition of nail marks at the autopsy 5 hours after removal of the corpse from the water and drying |
| Weiler and Riße [179] | Electrocution | Characteristically shaped electrical burn related to a tool employed by the perpetrator in his printworks |

*Source:* From Schmidt P and Madea B. *Forensic Sci Int* 1995;**72**:135–146.

manual strangulation, two by electrocution). The results of the toxicological analyses, including determination of BAC, are in contrast with the findings of Trübner and Püschel [169], whose cases of bathtub fatalities showed the highest percentage of people under the influence of alcohol among the victims of a homicide, but the results support the recommendation of Geertinger and Voigt [63] that negative toxicological examination should give special ground for suspicion of homicide.

Looking at the case reports, in Case 1 the autopsy findings were in good agreement with the later confession of the perpetrator. There is no doubt that, referring to Trübner and Püschel [169], the scenario and autopsy findings of this fatality were highly indicative of the manner and cause of death. According to the classification of Kruger and colleagues [90], the corpse had merely been deposited in the bathtub and this was of no importance for the occurrence of death. The motive for this behaviour could not be elucidated. In comparison, Devos et al. [42] reported a homosexual being strangled and then put into the bathtub to make the fatality appear non-homicidal. In another case the perpetrator explained that he had stabbed his wife and then given her a shower to remove the traces [90]. In a further case in our material, the perpetrators described stabbing their victim and then submerging them in the bathtub to ensure death.

In Case 2, the autopsy findings proved vital strangulation and agonal drowning. Considering the circumstances at the scene, the severe injuries of the neck as well as the haemorrhages in the skin and the soft tissues of the neck may have been related to a blunt force pushing the body against the front side of the bathtub, and they leave no doubt as to the diagnosis of homicide. Referring to the systematic approach of Kruger et al. [90], the scenario suggests that the homicide was committed in the bathtub and that the mechanism of death was related to the particular facilities.

In Case 3, the lethal strangulation was not discovered before the autopsy. Particularly in cases of ligature or manual strangulation, the police and the forensic pathologist may encounter serious difficulties in establishing the cause of death (as observed by Spitz [161]). Mueller [121] has emphasized that abrasions of the skin due to manual strangulation or other causes may be barely visible on wet skin but become more obvious some hours later. Madea et al. [108] demonstrated in a series of postmortem experiments that the intensity of ligature marks is markedly diminished by exposure to water. In particular, the surface pattern reflecting the nature of the ligature may disappear. Bode and Kampmann [28] succeeded in making an experimentally produced strangulation furrow completely disappear by combined treatment with ointments and exposure to water. Betz et al. [24] showed in an experimental model that petechial bleedings of the conjunctivae may disappear after 4 hours of freshwater-lodging, probably due to haemolysis caused by the hypo-osmolaric medium. Lasczkowski et al. [99] elucidated the particular diagnostic problems brought about by putrefaction of a corpse found in the bathtub.

These observations and experimental results underline the belief that a thorough autopsy should be carried out on every suspicious fatality in the bathtub, even if first examination at the scene does not reveal any severe injury.

## 18.6 Child homicide in Cologne (1985–1994)

### ■ *Peter Schmidt and Burkhard Madea*

Twenty-five child homicides investigated in the Cologne University Institute of Forensic Medicine from 1985 to 1994 were retrospectively analyzed with special reference to the evidential value of the autopsy findings and possible

**Table 18.7 Cause of death ($n = 33$)**

| Cause of death | Number |
|---|---|
| Blunt injury | 9 |
| Strangulation | 6 |
| Smothering | 3 |
| Drowning | 2 |
| Neglect (starvation) | 5 |
| Sharp violence | 6 |
| Gunshot | 2 |
| Combined injury | 7 |

*Source:* Schmidt P et al. *Forensic Sci Int* 1996;**79**:131–144. [147]

peculiarities related to the infant's physiological condition. About 65 per cent of the victims were boys younger than 3 years. About 65 per cent of the child homicides were committed by the mother in the parental flat, predominantly in the early hours of the evening during the weekend. The modes of death were 9 blunt injury, 6 sharp violence, 6 strangulation, 3 smothering, 2 drowning, 2 gunshot and 5 neglect (starvation) (Table 18.7). The defenseless, helpless and immobile condition of the infant particularly favours a homicide by manual assault, smothering or neglect (starvation).

The autopsy findings in the cases of lethal strangulation are given in Table 18.8. With the exception of one case with a deep incised wound of the neck, external injuries such as ligature mark, abrasions, contusions and fingernail marks were unequivocally visible, and the same was true for petechiae of the conjunctivae, sclerae and face. Haemorrhages of the soft tissues surrounding the thyroid cartilage and the hyoid bone were frequently encountered but, due to minor calcification in the child, no fracture was found. Our material comprises three additional asphyxic deaths due to smothering which showed few discrete abrasions in the vicinity of the mouth and the nose, very fine petechiae on the skin of the face and in the conjunctivae and spare Tardieu spots. In every case the autopsy diagnosis was confirmed by a confession of the perpetrator.

In the cases of homicidal strangulation, the death scene investigation had already indicated the nature of the fatality, so a distinction from suicidal [105] and especially accidental strangulation, did not need to be established.

**Table 18.8 Autopsy findings in homicidal strangulation**

| Form | External marks of violence | Petechiae | Hyoid/ thyroid | Soft tissue haemorrhage |
|---|---|---|---|---|
| Ligature | Present | Multiple | Haemorrhage | – |
| Manual | Present | Few | – | Severe |
| Manual | Present | Multiple | Haemorrhage | Severe |
| Manual | Present | Multiple | – | Severe |
| Manual | Present | – | Haemorrhage | Little |
| Manual | Discrete (incised wound) | Multiple | – | Moderate |

*Source:* Schmidt P et al. *Forensic Sci Int* 1996;**79**:131–144. [147]

The autopsy findings – in quite close agreement with the results of an analysis of 30 fatal strangulations in childhood including 18 homicides [103] – were without any doubt appropriate to support the conclusions drawn from the death scene. Nevertheless, it must be emphasized that, particularly in a child, a ligature mark may be barely visible or absent when the ligature is soft and removed immediately after death and little resistance is offered by the victim. In manual strangulation, external injuries of the neck may also be absent due to the discrepancy between the infant's thin neck and the large hand(s) of the perpetrator. Lack of petechial haemorrhages has been reported and is attributed to the assault on the weak infant's neck causing immediate complete arterial obstruction [46,69,103,145].

In the three cases of homicidal smothering/suffocation in our material, the diagnosis could be proved beyond doubt because the conclusions suggested by the autopsy findings were confirmed by respective confessions of the perpetrators. But it may be impossible to establish the diagnosis solely on the postmortem examination. The autopsy may fail to disclose any injury around the mouth and the nose or signs of asphyxic death when a soft pillow or the hands were used to smother an infant incapable of resistance. In addition, discrete petechiae of the face, sclerae, conjunctivae, epicardium and pleural surfaces are so non-specific that they do not allow a clear distinction from SIDS [45,54,55,159]. As a consequence, 2–10 per cent of unexpected and unexplained 'cot deaths' are estimated to be due to filicide [54,55], and several case reports of suffocation previously diagnosed as SIDS have been published [114]. Substantial contributions to the elucidation may be provided by a possible confession [119], detailed analysis of repeated apnoeic episodes in the same environment [47,55], careful investigation of the death scene [19], observations of witnesses [21,55] and psychosocial study of the family and its background [21,54]. Video surveillance initiated by recurrent cyanotic episodes in infants suspected to be due to smothering has shown that the heart rate starts to decrease 30 seconds after the obstruction of the airways and EEG slows and flattens at 90 seconds. As the victims were demonstrated to struggle violently, labelling of smothering as 'gentle' battering should be abandoned [142,159].

## 18.7 Simulation of a Homicide

■ *Burkhard Madea and Peter Schmidt*

A retrospective analysis of the autopsy material of the Institute of Forensic Medicine of the University of Bonn from 1992 to 1996 revealed 11 suicides of children and adolescents (8 girls and 3 boys). Their ages ranged from 10 to 19 years [182]. The suicides occurred preferentially outdoors in the warm months of summer, on a Monday or in the middle of the week, and in the afternoon or early evening. Independent of sex, the children unexceptionally applied hard suicide methods such as hanging or jumping from a height. Regarding the psychological background, prevailing factors were current conflicts within the family or at school and chronically disturbed family structures.

## Case report

In one case of the suicidal hanging of a 10-year-old girl, the mother tried to pretend that a homicide had been committed to conceal the child's suicide [183]. However, characteristic findings of the postmortem examination and conclusions from the analysis of forensic autopsy series provided substantial evidence for the police investigation.

In the late hours of a spring night the police authorities were informed that a 10-year-old girl had been found dead in a playground near her parents' house. Since injuries to her neck were noticed by the police officers, a homicide investigation was initiated. The postmortem examination showed a furrow slanting symmetrically from the front to the nape of the neck (Figure 18.14), discrete punctate haemorrhages of the skin of the face, and numerous 'tramline' bruises of the back, buttocks and extremities. There was no doubt that the 'tramline' bruises were the result of child abuse (Figure 18.15). Furthermore, at the autopsy, intervertebral disc haemorrhages of the lumbar spine (Simon's sign) were found. Haemorrhages of this kind are seen in cases of hanging but not of ligature strangulation. The findings indicated as cause and manner of death suicidal hanging following corporal punishment.

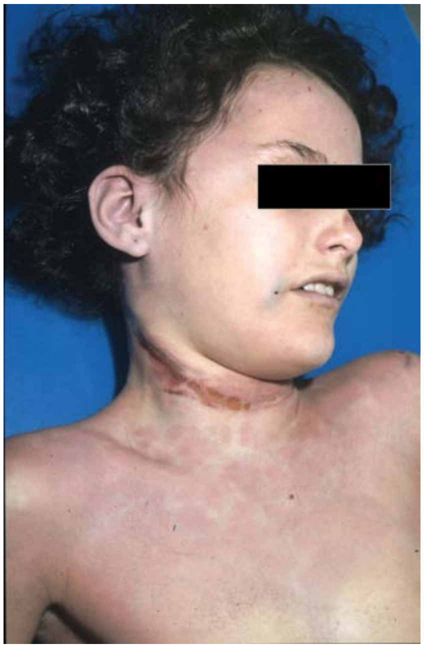

**Figure 18.14** Furrow slanting from the front to the nape of the neck, typical for hanging but not for ligature strangulation.

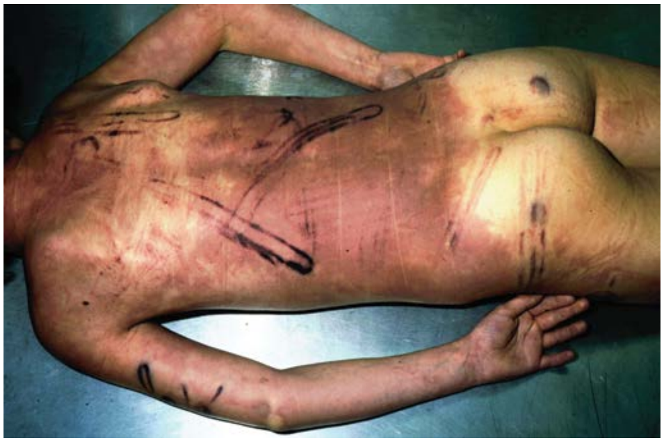

**Figure 18.15** Several 'tramline' bruises of the back, arms and buttocks.

Confronted with these conclusions, the mother immediately made a confession: she said that the child had stolen money from an aunt and was punished by the mother. The mother was later found dead. The cause of her death was suicidal hanging. A suicide note from the girl was also found. The mother had tried to pretend the death was a homicide to conceal the child abuse and prevent trouble with her husband.

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# 19 Suicide

*Frank Musshoff, Burkhard Madea and Elke Doberentz*

## 19.1 Fatality due to ingestion of tramadol alone

### *Frank Musshoff and Burkhard Madea*

Tramadol is a centrally acting analgesic used for the treatment of moderate to severe pain. The drug has a weak affinity for the μ-opioid receptor and inhibits the reuptake of norepinephrine and serotonin. Side effects from tramadol use include seizures and respiratory depression [46].

Recently, Michaud et al. reported a case of a 30-year-old woman with a history of depression who was found dead after the ingestion of alprazolam, tramadol and alcohol [59]. In the peripheral blood, alprazolam was detected at a concentration of 0.21 mg/l, the tramadol concentration was determined as 38.3 mg/l and the blood alcohol concentration was 1.29 g/kg. The tramadol concentration was more than 100 times the normal therapeutic range of 0.1–0.3 mg/l. Nevertheless, the authors could not ascribe the death solely to tramadol because of high levels of ethanol and alprazolam. Deaths attributed to tramadol intoxication alone have been described only rarely until now [28,47]. We know about one case concerning a 6-month-old child with a blood concentration of tramadol of 2 mg/l [79], a suicide by tramadol overdose with a whole blood concentration of 13 mg/l (additionally, 7-aminoflunitrazepam was determined in a concentration too low to have contributed to death) [50], and a tramadol overdose fatality with a blood concentration of 15.1 mg/l [60].

We report here a further case of a fatal intoxication in an adult with tramadol alone. In addition, we determined the tissue distribution of the drug.

### Case report

A 26-year-old male nurse who worked in an intensive care unit (body weight 70 kg, body length 191 cm) arrived at home at 6:30 a.m. from a night duty. He told his girlfriend, who was about to go to work, that he was tired and would go to bed. At 7.30 p.m., the woman found the man dead lying face down on the bed. Rigor mortis had already established. A bottle of Tramal® (tramadol) was found in his trouser pocket. One year before, the male nurse had consumed Rohypnol® (flunitrazepam), which he had stolen from the hospital.

External examination of the body revealed no signs of external violence. The autopsy and the histopathological examination revealed severe oedema of the brain (weight 1540 g) and lungs (left lung 1145 g, right lung 1135 g), congestion of all inner organs but no pre-existing diseases contributing to or as the cause of death.

### Toxicological analysis

Various body fluids and organ tissues were assayed for ethanol and drugs of abuse (acidic, basic and neutral organic drugs) using routine methods including immunochemical procedures and liquid–liquid as well as solid-phase extraction procedures with further analysis by gas chromatography/mass spectrometry (GC/MS) and high-performance liquid chromatography with diode array detection (HPLC/DAD). The latter was used for quantitative determination of tramadol.

Following standard extraction procedures, tramadol, *N*-desmethyltramadol and *O*-desmethyltramadol were identified in the urine sample by GC/MS by comparison of their mass spectra with reference library spectra and reference material. Quantitative analysis of tramadol was performed by HPLC/DAD. The results are shown in Table 19.1. All other tests for ethanol and drugs of abuse were negative. Neither flunitrazepam nor 7-aminoflunitrazepam was detected.

### Discussion

The peripheral blood concentration of tramadol was quantitated at 9.6 mg/l, which exceeded the normal therapeutic range by at least 30 times. The highest concentrations were measured in urine and bile. The concentration of tramadol in liver and kidney, in relation to blood, failed to suggest a major sequestration of drug in either specimen. This was consistent with the reported volume of distribution ($V_{ds}$) of 3 l/kg. The concentration of tramadol in the heart and peripheral blood specimens did not suggest a major difference (ratio of 1.36) and this, too, was consistent with the drug's lack of sequestration in the liver. However, the findings were similar to those of morphine fatalities ($V_{ds} = 3.3$ l/kg) [15,48]. Also similar to morphine, tramadol was accumulated significantly in the bile. A hair sample was not collected, so we were unable to look for a possible chronic administration of tramadol in the present case.

Table 19.1 **Tissue distribution of tramadol in the present case**

| Specimen | Tramadol |
|---|---|
| Urine (mg/l) | 46.0 |
| Heart blood (mg/l) | 13.1 |
| Femoral blood (mg/l) | 9.6 |
| Liver (mg/kg) | 6.2 |
| Bile (mg/l) | 46.1 |
| Kidney (mg/kg) | 3.1 |
| Gastric contents (not quantified) | Positive |

Considering the autopsy findings, the results of toxicological analysis were consistent with the assumption of a fatal overdose due to tramadol alone. Respiratory depression may be assumed to be the underlying pathophysiological mechanism. Our results support the observations that a high dosage of tramadol may lead to death even in the absence of interacting drugs.

## 19.2 Strangulation: Suicide at the wheel

■ *Burkhard Madea and Elke Doberentz*

Suicide at the wheel is a common and well-known problem in forensic medicine. In the past, several authors have suggested that some road traffic crashes were actually suicide attempts [32,33,35,42,54,76]. However, it is very difficult to identify cases of driver suicide among motor vehicle crashes since the suicidal intent is not as self-evident in such cases as it is, for example, in cases of hanging. Meanwhile, a number of risk factors for driver-assisted suicides have been identified (Table 19.2) [35].

Other modes of suicide also use cars, such as vehicle-assisted strangulation, decapitation and CO poisoning [7,8,13,14,22,23,33–35,53,57,85,92].

Table 19.2 **Risk factors associated with driver suicides* (according to Henderson and Joseph [35])**

- Males
- Age between 25 and 34
- Single-occupant crashes
- Non-wearing of seat belts
- Head-on collision
- Single vehicle crash (into a tree or pole)
- Collision into a heavy goods vehicle
- Absence of skid marks or other evidence for loss of control over the vehicle
- Alcohol intoxication/abuse
- Significant recent psychological stress
- Mental disorders (such as depression) and previous suicide attempt(s)
- Impulsivity and low distress tolerance personality trait

* Reprinted from Henderson and Joseph [35], with permission from Elsevier.

While in some cases the car is used to commit suicide through velocity, or in conjunction with fuel or gases, in other cases a vehicle just represents the location of the suicide as it could have been committed at any other place (Table 19.3). Some authors have recommended classifications of automobile-related suicides (Tables 19.4 and 19.5) [14,33].

Cases of strangulation, hanging in the car using a rope or even suicidal strangulation by a vehicle seatbelt have been reported [14,23,33,53]. In recent years, reports of several cases of motor vehicle-assisted ligature strangulation causing complete decapitation have been published [7,13,22,34,57,85,92].

Three cases of suicidal strangulation in a car or using a car for suicide are described here. In two cases, homicide was initially assumed and, in Case 1 particularly, extensive police investigations were necessary.

Table 19.3 **Suicide at the wheel**

| Vehicle used for suicide | Vehicle just location of suicide |
|---|---|
| Vehicle-assisted decapitation | Intoxication |
| CO poisoning | Burning |
| Suicidal traffic crash | Strangulation |
| Suicidal burning (car fire) | Shooting |

Table 19.4 **Classification of automobile-related suicides (according to Hardwicke et al. [33])**

| Multiple injuries | Drowning | Carbon monoxide poisoning |
|---|---|---|
| Head-on collision of a single occupant vehicle with a fixed roadside object: no skid marks, accelerating imprints on shoe soles<br>Falls from height | Falls from height (bridges or cliffs) into water | Subject sitting or lying in an intact vehicle parked in a closed garage with engine running |

Table 19.5 **Classification of automobile-related suicides: suicide with motor vehicle as integral part of the process (according to Byard and James [14])**

Cases fell into seven categories where the deceased individuals had used:

- Vehicle exhaust to cause fatal carbon monoxide toxicity
- The speed and mass of their own vehicle to inflict lethal injuries, i.e. driving a vehicle into a stationary object at speed, or driving over a cliff
- Speed and mass of another vehicle to sustain lethal injuries, i.e. stopping in front of a large vehicle moving at speed, stopping a car on train tracks
- The weight of vehicle to assist drowning
- Vehicle inflammability to assist self-immolation
- Vehicle fixtures (e.g. seatbelts) or ropes for hanging and ligature strangulation
- The vehicle to set up a crash situation as a back-up to suicide by other means

## Case 1

A 27-year-old male was found dead sitting on the front driver seat of his car (Figures 19.1 and 19.2) with both arms beside his trunk. The car was closed and locked. Around the neck and headrest there was a 79 cm long and 4 mm diameter plastic-coated rope that was knotted at the back of the neck behind the headrest. In the knot, there was a 24.5 cm long metal stick that had obviously been used to twist the rope tightly (Figure 19.3).The stick had been twisted four times, thus producing a second knot. The man was normally clothed. The temperature in the car was 13.7°C, deep rectal temperature was 29.5°C, and postmortem interval was about 6 ± 2.8 hours. The man was known to have had connections to the local drug scene and to have been drug-dependent some years before. He had obviously been living in his car. He had lost much body weight during the previous months and had mentioned that he might commit suicide.

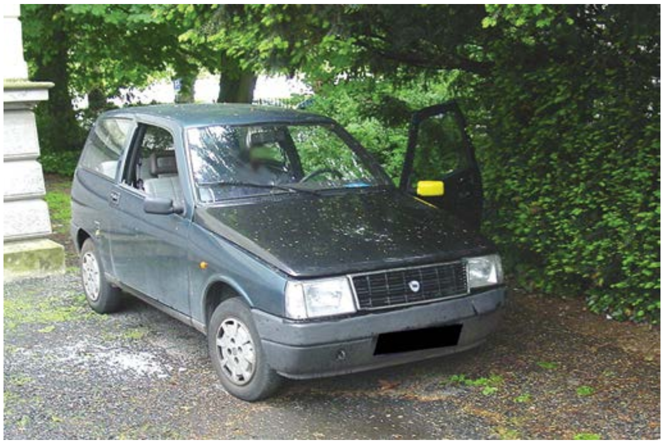
[redacted]

**Figure 19.1** Position of the car.

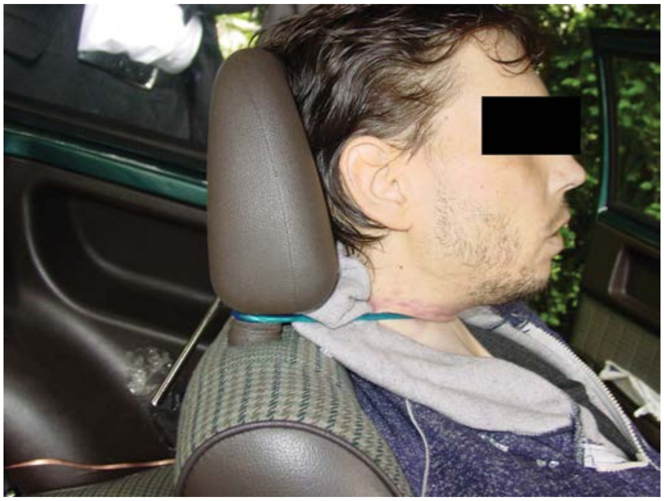
[redacted]

**Figure 19.2** Position of the deceased on the front seat with rope around the neck.

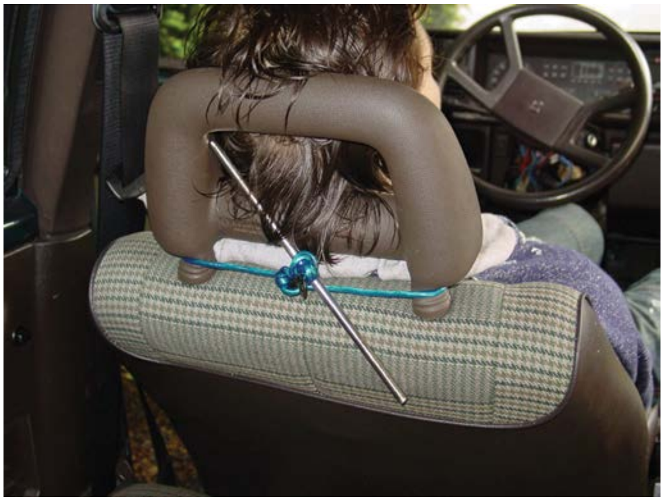

**Figure 19.3** Rope behind the headrest and metal stick within the knot.

### Main autopsy findings

- Body of a 27-year-old man.
- Body height 177 cm, body weight 72.4 kg.
- Corresponding to the coated electrical rope, a deep 4 mm wide strangulation mark at the front of the neck.
- Small superficial haemorrhages of the right sternocleidomastoid muscle at its front aspect.
- Small haemorrhage of the thyroid capsule.
- Haemorrhages of the conjunctivae.
- No other signs of violence, especially no indication of defence injuries.
- Cause of death: Suicidal ligature strangulation.
- BAC: 0.01‰.
- Toxicology: Negative.

A reconstruction revealed that a ligature strangulation by his own hand with the stick used to twist the rope was clearly possible (Figure 19.4).

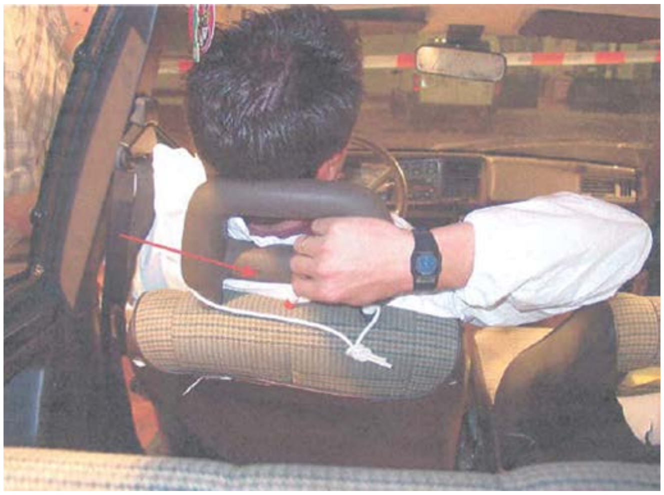

**Figure 19.4** Reconstruction established that self-strangulation was possible.

## Case 2

A 67-year-old man was found dead on the passenger front seat of his car (Figures 19.5 and 19.6). The car was closed and had been seen the day before in a parking place at a motorway. The man was normally clothed. Around the neck was a rope, the highest point of which was at the level of the right ear and the back of the neck (Figure 19.7). The rope was fixed to the handle of the passenger front seat with two knots (Figure 19.6). Corresponding to the rope, a strangulation mark ascending from left to right was seen.

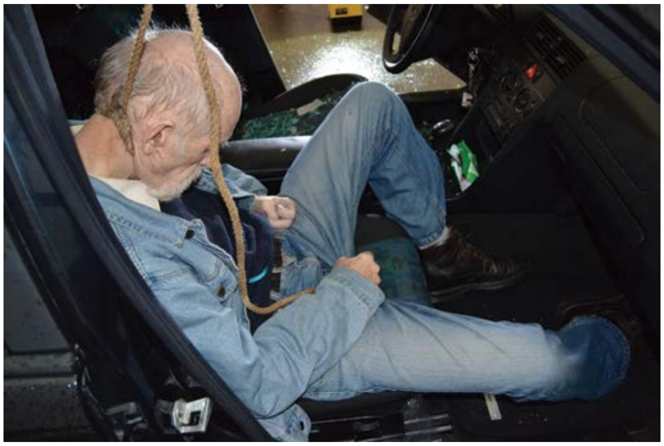

**Figure 19.5** Position of the man on the front passenger seat.

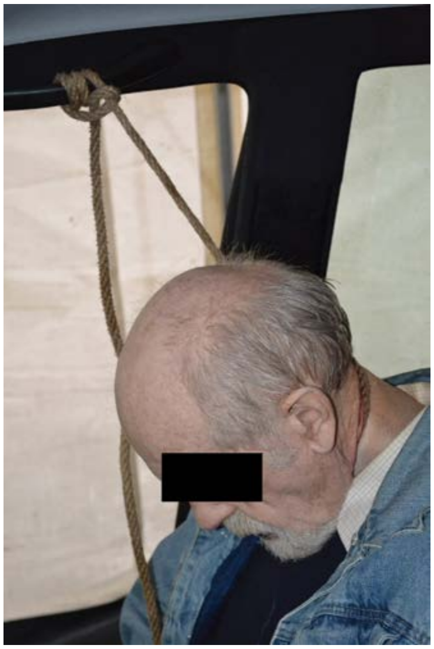

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**Figure 19.6** Rope fixed on the grip of the passenger front seat with two knots.

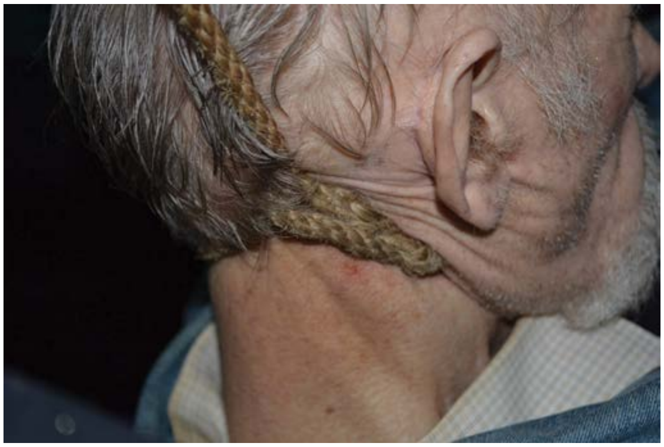

**Figure 19.7** Knot behind the right ear.

## Main autopsy findings

- Body of a 67-year-old man.
- Body height 181 cm, body weight 75.2 kg.
- Strangulation marks on both sides of the neck with the highest point of the strangulation mark behind the right ear.
- Strangulation mark corresponding to the used rope.
- Petechiae of the eyelids and the conjunctivae.
- Small haemorrhage within the front neck muscles.
- Haemorrhage of the left upper superior thyroid horn.
- Pulmonary and brain oedema.
- Ponderable coronary atherosclerosis.
- Cause of death: Suicidal hanging.
- BAC: 0.00‰.
- Toxicology: Negative.

## Case 3

A 48-year-old man was found in front of a cemetery in a supine position beside his car (Figures 19.8 and 19.9). The

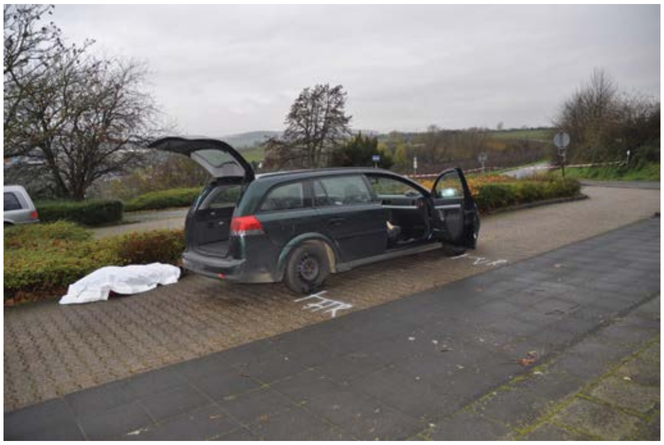

**Figure 19.8** Position of the car in front of a cemetery, the covered deceased behind the car.

engine of the car was running and the driver's door was open. Around the neck there was a rope with a hangman's knot (Figures 19.10 and 19.11), corresponding to the strangulation mark. Fifty metres away, a 10 metre long rope was found fixed to a tree, with its end corresponding to the rope around the man's neck (Figure 19.12).

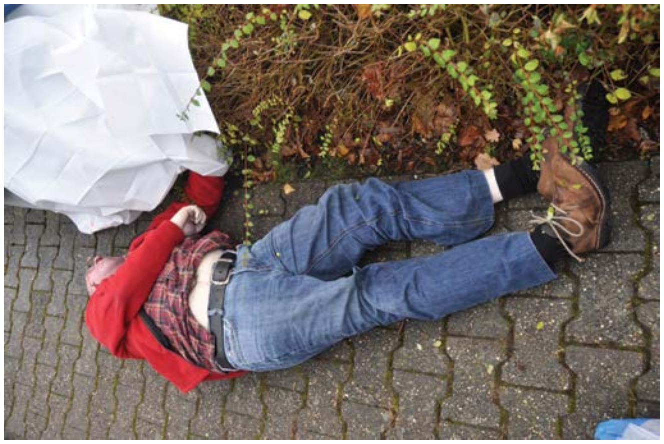

**Figure 19.9** Position of the deceased.

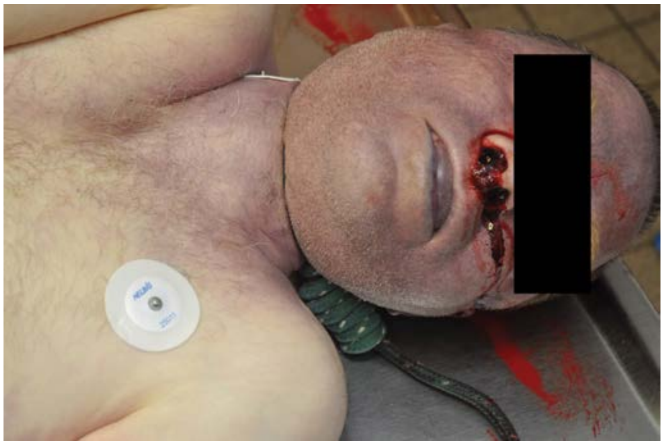

**Figure 19.10** Marked congestion of face, protrusion of the tongue, and bleeding from the nose due to mucosal congestion.

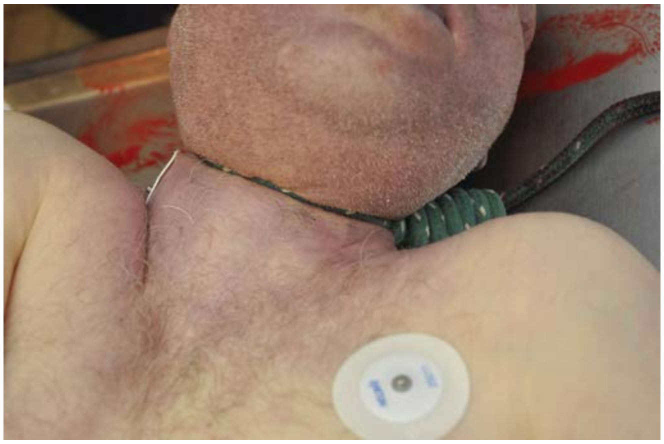

**Figure 19.11** Knot on the left side of the neck.

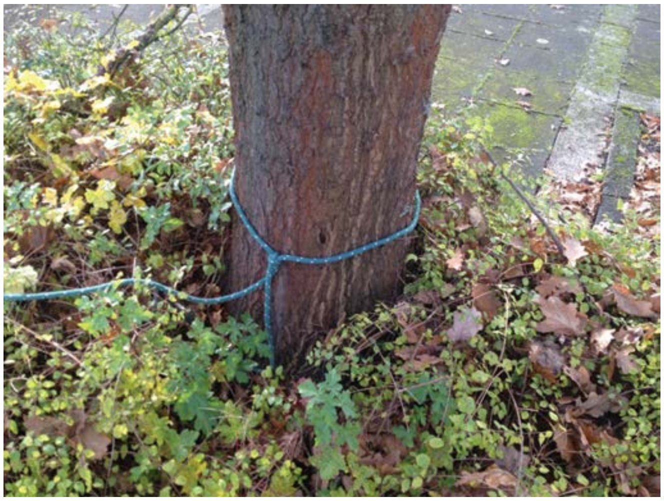

**Figure 19.12** Rope fixed at a tree 50 metres away from the position of the man.

### Main autopsy findings

- Body of a 48-year-old man.
- Body height 170 cm, body weight 80 kg.
- Strangulation mark on the neck corresponding to the rope.
- Massive congestion of the head and face with intense petechiae of the conjunctivae and eyelids.
- Haemorrhage of the left sternocleidomastoid muscle.
- Fracture of the right superior thyroid horn.
- Small haemorrhages of the tongue.
- Brain oedema.
- No other injuries, especially no indication of defence injuries.
- Cause of death: Suicidal strangulation.
- BAC: 0.49‰.

This was obviously a case of attempted vehicle-assisted ligature strangulation, but the rope had disrupted and the man had been pulled outside the car, since the driver's door was open. Homicidal ligature strangulation with the rope, as it was found around the neck of the male, was deemed to be impossible.

## Discussion

At first view, homicide was assumed in Cases 1 and 3. In Case 2, a suicide was directly obvious although the car was not found at a typical site for suicidal hanging. In Case 3, the police did not realize during the first examination of the scene of death that part of the rope was fixed to the tree 50 metres away from the position of the corpse. Therefore, suspicion of homicidal ligature strangulation was raised. At autopsy, a hint was given to the police that they should look for the missing part of the rope because the case had been presented as a 'typical' vehicle-assisted suicide.

In Case 1, the reconstruction revealed that suicidal ligature strangulation was possible and further typical findings of suicidal ligature strangulation (several knots, twisting of the rope using a stick) were present [9,41,44,52,55].

Motor vehicles have become important instruments of self-destruction [14] and even unusual suicides involving motor vehicles are known.

Forensic pathologists must be aware of these rare suicides to prevent unnecessary investigations but so must police officers. In addition, a thorough examination of the death scene and its surroundings will avoid overlooking important findings which may be essential to reconstruct the course of death. In Case 3, for example, the police were at first convinced they had to deal with a homicide but this could easily have been obviated if the *whole* of the death scene had been thoroughly examined initially.

## 19.3 Combined suicide by oxydemeton-methyl (Metasystox®) ingestion and hanging

■ *Burkhard Madea and Frank Musshoff*

### ■ Case report

The body of a 39-year-old man without any history of drug abuse or illness was found in a civic storage building, hanging on the cage of an elevating work platform (cherry picker). The switching panel of the elevator showed signs of ‘cage operation’ (Figure 19.13). Preceding short messages and telephone calls suggested a suicide. Among other items, an open and almost empty bottle of Metasystox®, a mobile (cell) phone and an empty bottle of beer were found in the cage (Figure 19.14). A residual amount of insecticide was found in the bottle. As the bottle seemed to be of older origin, it was not impossible that traces of the insecticide were present due to its original purpose and use. Thus, it was not possible to know the total amount of ingestion.

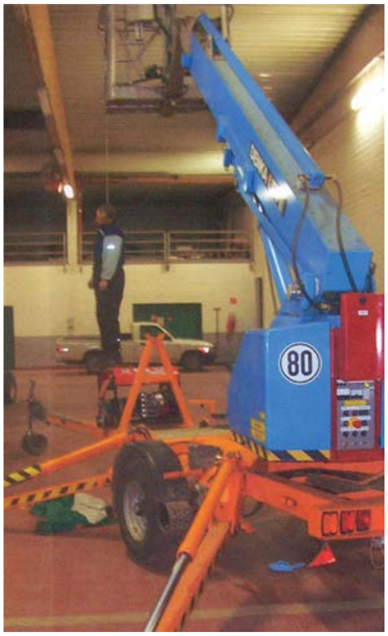

**Figure 19.13** Crime scene.

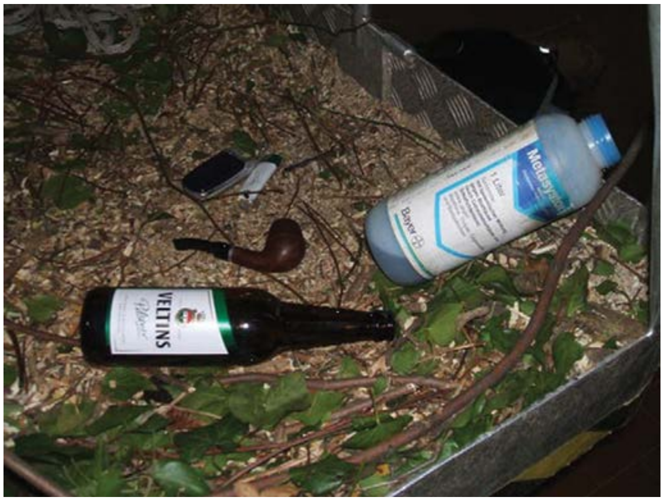

**Figure 19.14** Findings near the body.

### Main autopsy findings regarding strangulation

- Strangulation rope with corresponding strangulation mark, rising from the front to the back of the neck (Figure 19.15).
- Fracture of the left superior thyroid horn with surrounding haemorrhage.

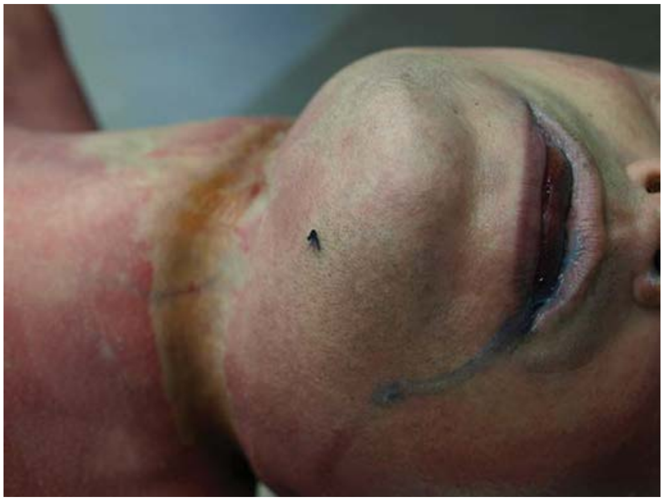

**Figure 19.15** Strangulation mark; blue salivation.

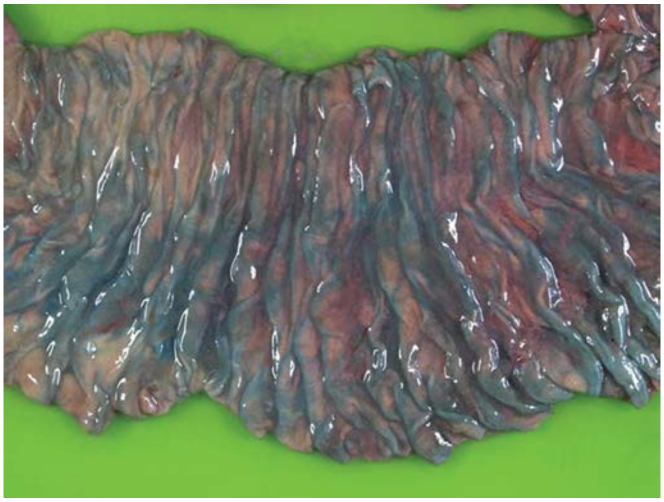

**Figure 19.16** Bluish contents in the duodenum.

- Fluidity of cadaveric blood.
- Hypostasis of all inner organs corresponding to the hanging position.

### Main autopsy findings regarding poisoning

- Blue salivation of the left angle of the mouth, as well as a blue adherence on tongue and oesophagus.
- Bluish content of the stomach, similar bluish content also in the duodenum and jejunum (Figure 19.16).
- Brain oedema.
- Pulmonary oedema.

### Discussion

The transportation distance of the bluish content into the small bowel led to the question of whether the victim had survived the intoxication for an unusually long time and the strangulation took place after a longer time interval.

A case of a combined suicide by oxydemeton-methyl (Metasystox®) ingestion and hanging was reported. Dimethyl phosphate (DMP) proved to be a stable product of oxydemeton-methyl; for its determination a gas chromatographic/mass spectrometric procedure was performed. DMP levels were quantified in several liquids and tissue samples: measured concentrations were 103.31 mg/ml DMP in gastric contents, 0.10 mg/ml in blood, 1.40 mg/g in liver, 2.87 mg/ml in bile and 0.80 mg/g in kidney. No DMP was found in either urine or cerebrum. On the basis of the distribution of the poison in the body, and particularly due to the fact that no DMP could be detected in the urine and also that DMP was found in the gastric contents, it can be assumed that there was a close temporal connection between ingestion of poison and hanging. Therefore, this was a case of a primary combined suicide.

## 19.4 Report of an unusual case of hanging in a lying position

### *Elke Doberentz and Burkhard Madea*

Hanging is still the most common suicide method among men and women, even in high age groups. There are many variations of hanging. When considered in relation to the body weight, only a relatively small force is necessary to obstruct the carotid arteries. In this way hanging in a sitting, kneeling or even lying position is possible. An exceptional case of hanging in a lying position is presented here.

A 99-year-old, multi-morbid man (body height 159 cm, body weight 47 kg,) was found lying strangled in his hospital bed (Figure 19.17). The ligature used was a stiff spiral cable for remote control of the bed, which was directly attached to the wall behind its head end (Figure 19.18). The position allowed compression of the neck vessels, which resulted in marked congestion of the head, numerous petechial and confluent haemorrhages in the facial skin as well as blood loss from both ear canals (Figures 19.19 and 19.20). In the cervical soft tissue above the ligature, bleeding into the muscles and connective tissue as well as multiple haemorrhages in the tongue were found. Reconstruction at the scene showed that suicide was possible. In such unusual cases, extensive police investigations, forensic autopsy and reconstruction of the course of the event to prove feasibility are essential.

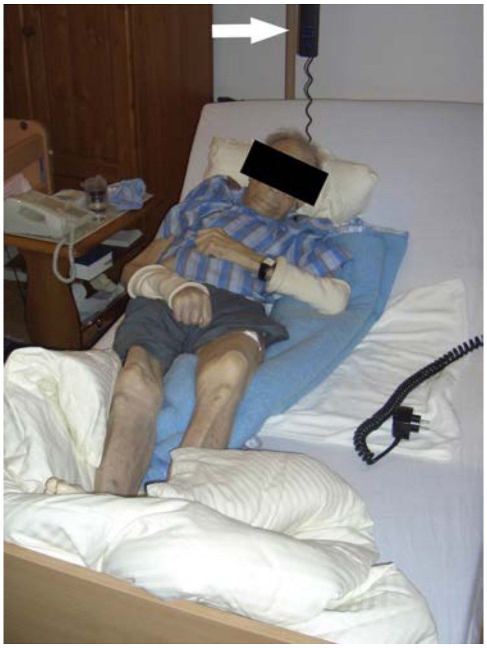

**Figure 19.17** Position at the scene of death, lying in bed in a dorsal position, the headboard of which has been put up rather high. Above the bed, the holder for the remote control of the bed (arrow) mounted on the wall, showing a linked helix cable.

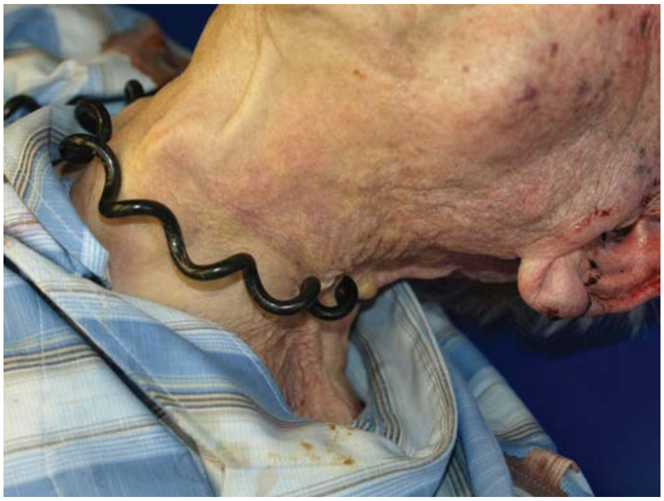

**Figure 19.18** Rigid helix cable used as strangulation tool.

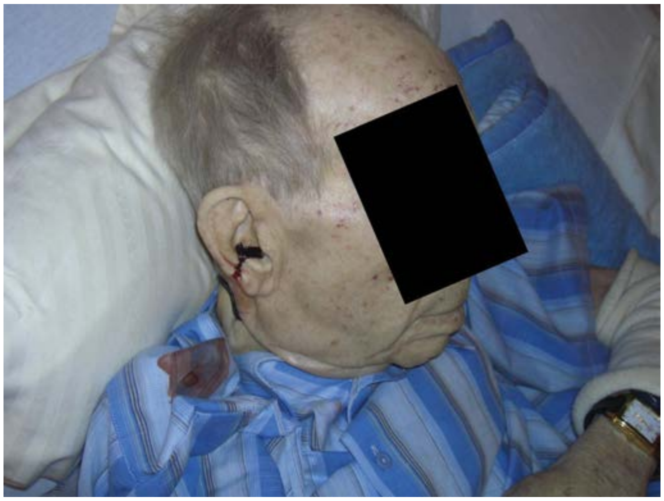

**Figure 19.19** Focal extravasations in the facial skin and from the ear.

## 19.5 Self-strangulation with two connected cable ties

■ *Elke Doberentz and Burkhard Madea*

Ligature strangulation is a fatal compression of the neck by means of a strangulation device, which is usually tightened using the hands or, in rare cases, by a machine. Most cases of ligature strangulation are homicides but suicides by ligature strangulation also occur. The strangulation device in cases of suicide is still in place and is wound around the neck several times. Furthermore, it may be knotted at the front of the neck.

In rare cases, cable ties are used for ligature strangulation, in both homicide and suicide cases. Figures 19.21–19.26 show the typical findings in a suicidal ligature strangulation with two connected cable ties.

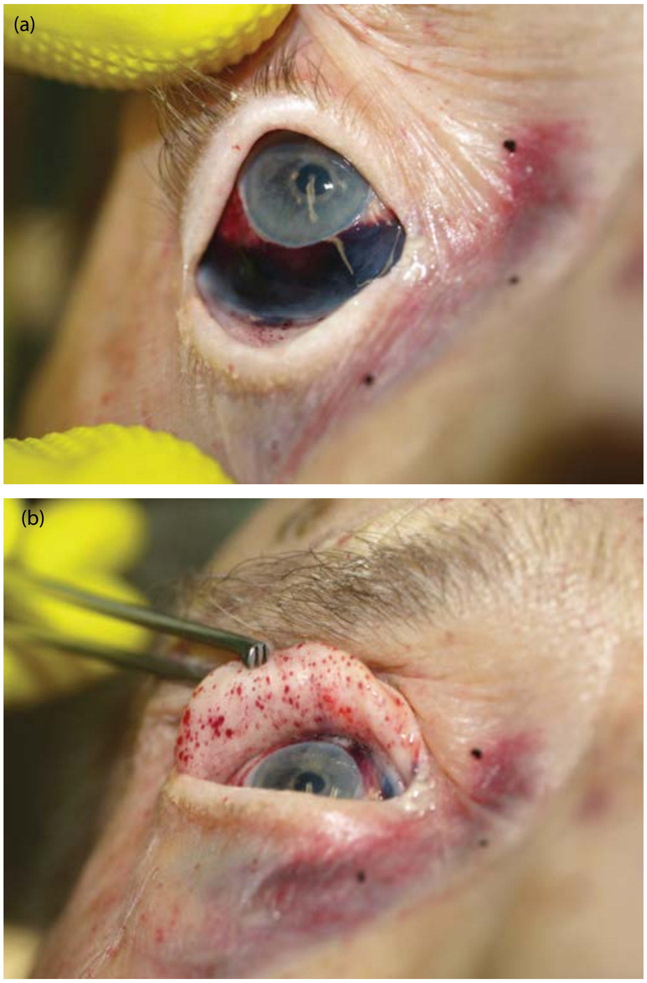

**Figure 19.20** (a), (b) Larger bleedings and petechial haemorrhages in the conjunctivae.

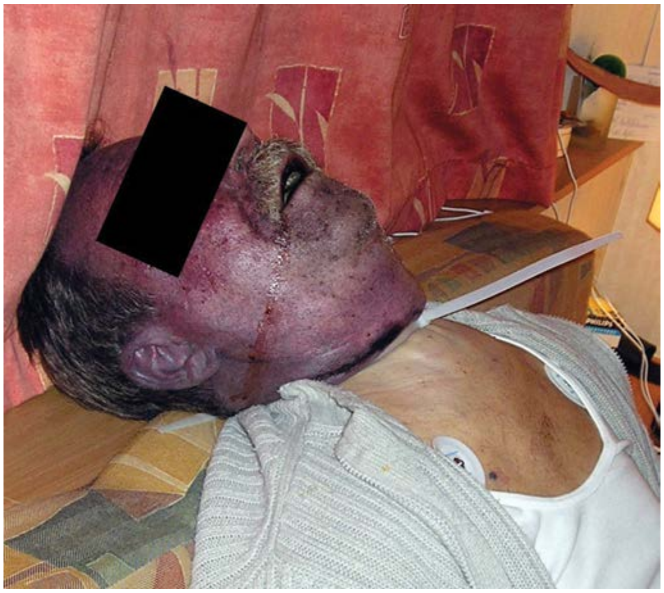

**Figure 19.21** Finding position with cable ties around the neck and congestion above the cable ties.

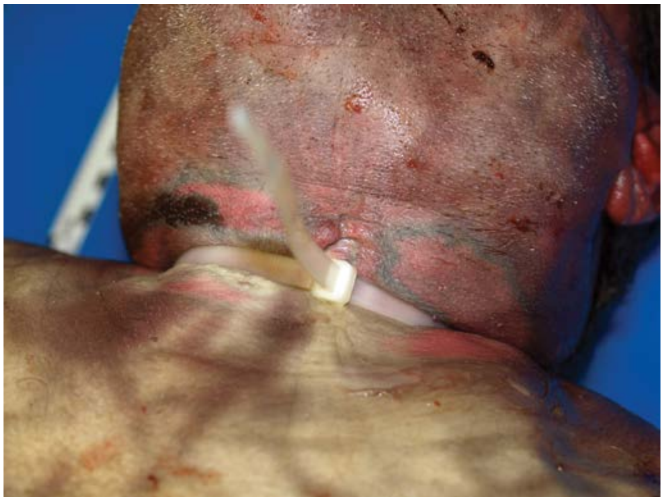

**Figure 19.22** Cable tie in front of the neck.

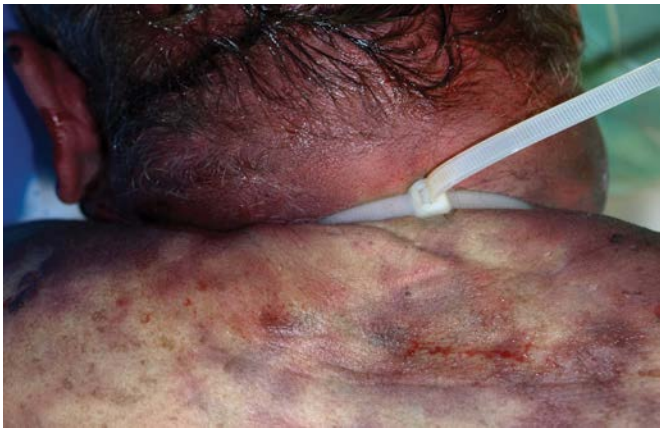

**Figure 19.23** Cable tie at the back of the neck.

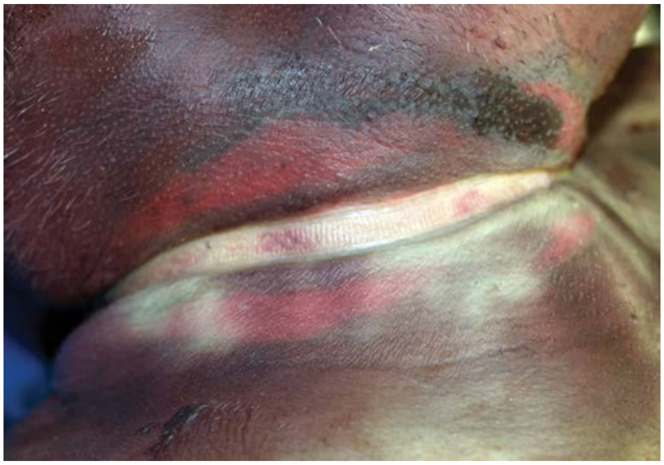

**Figure 19.24** Ligature mark.

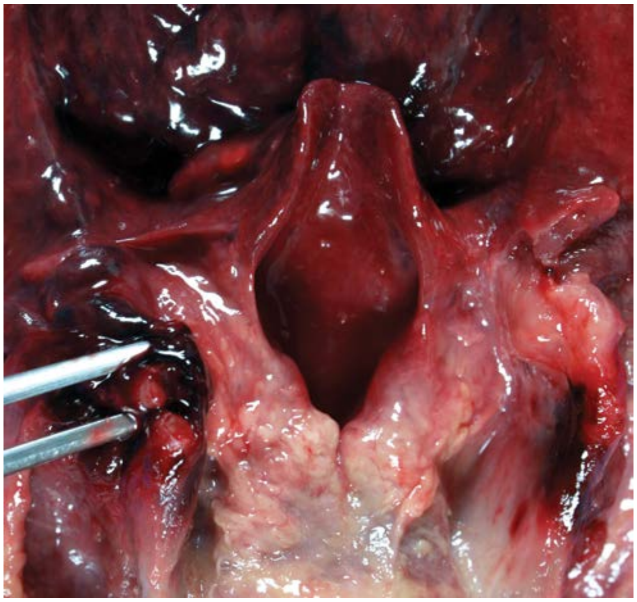

**Figure 19.25** Fracture of the left superior horn of thyroid cartilage with surrounding haemorrhage.

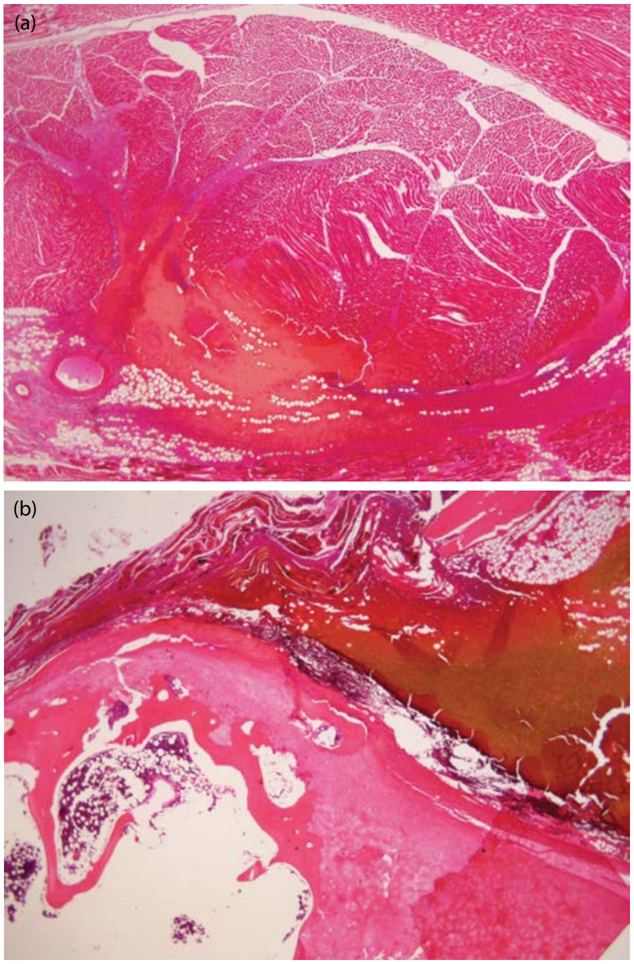

**Figure 19.26** (a) Haemorrhage of the tongue (H&E, ×40); (b) haemorrhage around the fracture of the left upper thyroid cartilage (H&E, ×40) without inflammation.

## 19.6 Two cases of suicide by asphyxiation

### *Frank Musshoff and Burkhard Madea*

Cases of suicide by asphyxiation with a plastic bag placed over the head, or other kinds of confined space such as motorcycle helmets or prepared filter masks, have been reported in the forensic literature over several decades [88,69]. An auxiliary method is the channelling of gases into a plastic bag, leading to reduction or replacement of oxygen in the inhaled air and therefore accelerating death by suffocation. In 2002, Ogden and Wooten [65] described the first case of suicide by helium asphyxiation. Since that time, and in addition to an accidental case [98], numerous suicides by helium asphyxiation have been reported worldwide. Unfortunately, in such case reports detailed toxicological findings were missing and the causes of death could only be determined by the circumstances of the cases [26,27,30,63–64,84]. In 2012, Bittorf et al. [5] reported four cases of helium-associated suicides, in two of which a determination of helium was successful. In a previously published review article, Howard et al. [36] summarized in detail several cases from the literature together with cases from North Carolina. Toxicological findings following an asphyxial suicide with helium in a plastic bag were described by Auwärter et al. [2], who also previously reported analytical investigations in a case of death by suffocation in an argon atmosphere [3]. Schaff et al. [78] demonstrated a procedure combining gas chromatography with thermal conductive detection for the analysis of helium in postmortem blood and tissue specimens. Suicides by use of argon have not been described until now, but argon is well known for euthanasia in animals [71,87]. Reports have been published of accidental argon fatalities in a hospital [89] and also recently a fatality due to argon gas embolism during prostate cryosurgery [77]. Two cases of suicide by asphyxiation due to helium and argon are briefly reported here.

### Case 1

On her thirtieth birthday a female was found by her mother lying on her bed. A plastic bag was over her head, corded around the neck. A flexible tube inside the bag was connected to a helium bottle belonging to a commercially available helium balloon kit found in the bedroom. The hands of the deceased were bonded behind her back with a rope. A suicide note was found together with a testament.

#### Main autopsy findings

- Body of an adipose female.
- Body height 167.5 cm, body weight 92.8 kg.
- No signs of external violence.
- Beginning putrefaction with green discolouration, slippage of skin and marbling.
- Advanced putrefaction of the internal organs.
- Liver steatosis.
- Splenomegaly.
- Results obtained by chemical–toxicological routine analyses using immunological as well as GC/MS and high-performance liquid chromatographic–mass spectrometric screening procedures: blood alcohol concentration 0.35 g/l, diazepam 0.10 mg/l and nordiazepam 0.04 mg/l (in femoral blood).

### Case 2

A 43-year-old man was found lying dead in the basement of his home. A plastic bag was placed over his head and corded around the neck. A flexible tube inside the bag was connected to an argon bottle. In addition, a bottle of chloroform was found in the room as well as instructions for how to commit suicide written by an Australian physician.

#### Main autopsy findings

- Body of a male.
- Body height 166.5 cm, body weight 79 kg.
- No signs of external violence.
- Petechial haemorrhages in the conjunctivae, Tardieu spots, congestion of internal organs.
- Splenomegaly.
- Brain oedema.
- No relevant pre-existing diseases.
- Apart from a citalopram concentration of 0.018 mg/l, chemical–toxicological routine analyses revealed negative results including for chloroform.

### Collection and analysis of gas samples

For the collection of gas samples from the lungs, the procedure described by Auwärter et al. [2] was modified as follows.

Each lung was collected in a separate plastic pail filled with water except for a very small volume. The pail was inverted and plunged into a basin of water and the residual air was sucked out of the pail and discarded using a syringe fitted with a T-piece. The lung was then manipulated with scissors and compressed by hand so that air/gases escaping from the lungs were collected in the top of the inverted pail. The gas volumes released from the lungs were collected with the syringe and directly pressed into a 20 ml-headspace vial that had been completely filled with water and crimped closely under water beforehand. For this purpose, the septum of the vial cap was punctured with a second cannula, while holding the vial with the bottom up and filling in the gas

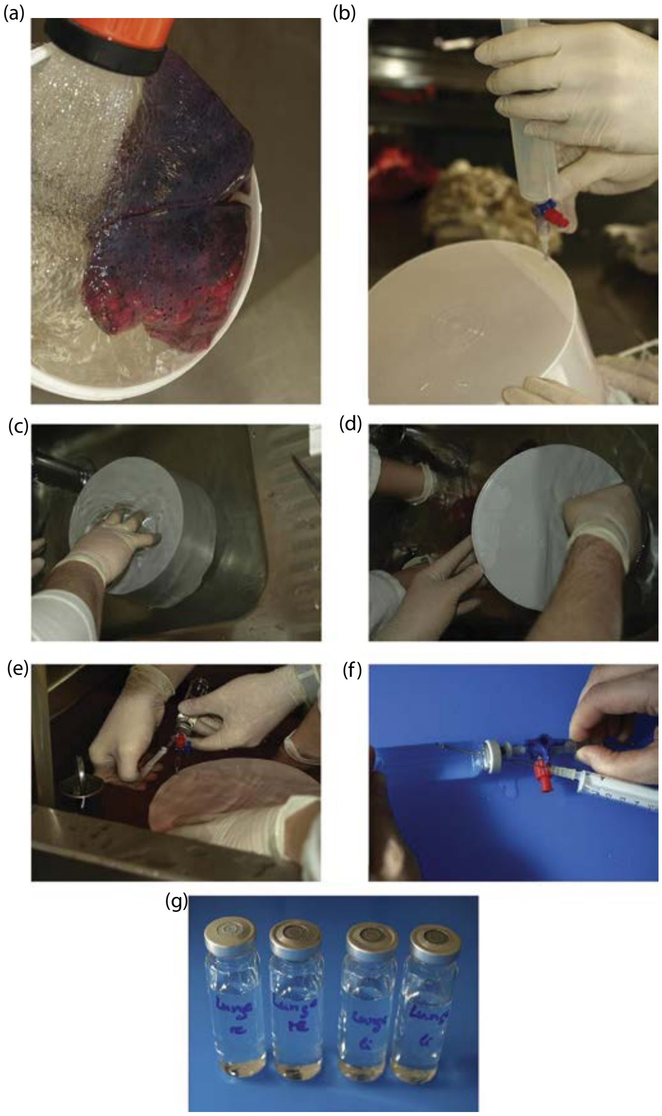

**Figure 19.27** Stages of the lung gas sampling procedure (a)–(g).

sample via the septum, so that the water was displaced through the second cannula. The stages of the procedure are shown in Figure 19.27.

Figure 19.28 shows the GC/MS chromatograms of gas samples obtained from the deceased in Case 1, from a comparison case without premortal helium exposure and from helium directly filled into a headspace vial. The standard revealed a positive result, but in the case under investigation as well as in the case without premortal helium inhalation, negative results were achieved. Figure 19.29 shows that positive results were obtained in the argon case (Case 2), with a significant peak for argon in comparison to the negative autopsy case.

## Discussion

The use of headspace GC/MS enables the detection in principle of inert gases such as argon and helium.

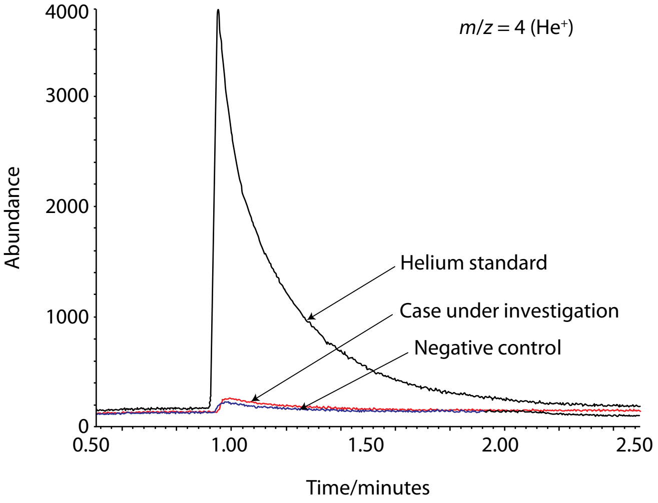

**Figure 19.28** Asphyxiation Case 1: The helium case. Headspace GC/MS-SIM chromatograms from the lungs of the deceased, from a comparison case without premortal helium exposure (negative control), and from helium directly filled into a headspace vial (helium standard). *m/z* = mass to charge ratio.

The approach is qualitative and there is no secondary confirmation method employed, but in combination with the observations and items found at the scene, this should provide enough information for a cause of death ruling. Important for this are an appropriate sampling technique at autopsy and a convenient analytical procedure (e.g. GC/MS analysis using nitrogen as the carrier gas). However, a number of factors may influence the results, such as a longer period of time between death and sampling or pre-analytical artefacts when sampling such highly volatile

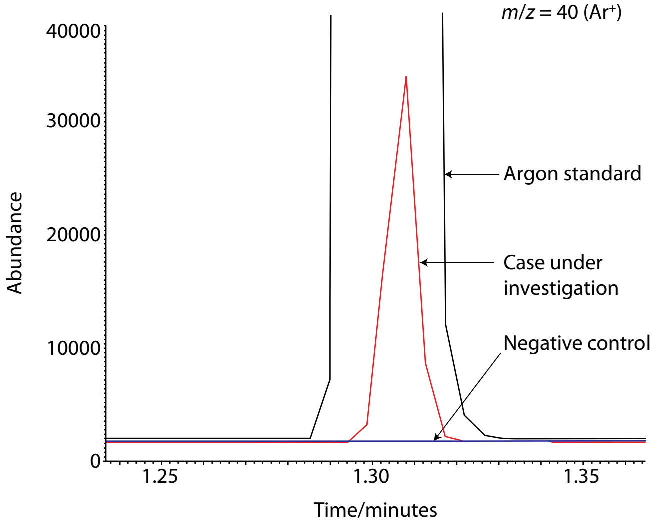

**Figure 19.29** Asphyxiation Case 2: The argon case. Headspace GC/MS-SIM chromatograms from the lungs of the deceased, from a comparison case without premortal argon exposure (negative control), and from argon directly filled into a headspace vial (helium standard). *m/z* = mass to charge ratio.

substances. Even in Case 1 involving helium presented here, only negative results were obtained despite the use of a potentially appropriate sampling technique. We cannot exclude a loss of helium by sampling in Case 1 because we did not have an internal standard for a definite proof. The postmortem interval until sampling was about 6–7 days in this case and putrefaction was observed which could probably influence further analyses. However, the scene of death gave clear indications and in both cases the cause of death was attributable to asphyxiation (in Case 1 in the absence of analytical data without a proof for helium inhalation), caused by environmental hypoxia. The manner of death was designated as suicide.

In general, classic but non-specific signs of asphyxia include petechial haemorrhages, as seen here in Case 2, congestion and oedema of the lungs, cyanosis, dilatation of the right ventricle of the heart, and fluidity of blood [43]. Plastic bag suffocation does not generally leave externally visible marks. The only possible indicators are certain spaces in the livores or merely an impression or a ligature mark on the skin caused by tight fixation of the plastic bag around the neck [31]. Petechial haemorrhages in the conjunctivae or the mucosa of the mouth are rare findings. We agree with Jones et al. [40], who emphasized that in cases of plastic bag suffocation an autopsy is not complete until toxicological analyses have been performed. Since positive morphological evidence is usually absent, investigations at the scene of death and the deceased's environment become extremely important. The cause of death can be determined only by morphological evidence at the scene of death and further detailed toxicological analyses.

In published suicide guides [37,39] and sites on the internet [66], insufflation of an inert gas into a suffocating plastic bag is recommended as a means to hasten death, and it has been reported that the number of such suicides has clearly increased over recent years [1,36]. The cases presented here occurred within 2 months.

In general, caution should be exercised because in some so-called suicide forums on the internet relatives are encouraged to remove used equipment such as helium cylinders, plastic bags and flexible tubes from the scene of death. This could conceal suicide for insurance/legal reasons and, in addition, would afford more dignity to the discovery of the deceased. From the forensic point of view, it must be acknowledged that such cases would probably remain unclarified because the sophisticated sampling needed (e.g. in airtight vials) would never be carried out.

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# 20 Accident

*Peter Schmidt and Burkhard Madea*

## 20.1 Death in the bathtub involving children

### *Peter Schmidt and Burkhard Madea*

Available studies dealing with ‘death in the bath’ provide only brief information on the peculiarities of children’s fatalities in the bathtub. A series of 42 deaths in the bath in Copenhagen and Gothenburg [1] included no children. A retrospective survey of 36 fatalities in the bathtub in Ghent [2] comprised 9 cases involving children. The causes of death were carbon monoxide intoxication, drowning due to lack of supervision and one homicidal immersion. Retrospective analysis of fatalities in the bathtub in Hamburg [3] revealed 24 cases in the age group ≤20 years (20 accidents, 2 homicides committed by the parents, 1 natural death and 1 undetermined case in connection with maltreatment). In a review of 46 deaths in the bathtub in Restock [4], 3 homicides of children were reported. (Data from the literature [2–16] are summed up in Table 20.1.)

The medicolegal significance suggested by these brief figures is underlined by the following data. In the United States, 710 persons drowned in bathtubs in 1979 and 1980, and children <5 years old accounted for 182 (25%) of these deaths [7]. Furthermore, in the years 1979–1982, in the United States 95 persons were electrocuted in bathtubs, with children <5 years having the greatest mortality rate of 3.7 per 10 million population per year [6]. Deliberate submersion of a child under water, particularly in the domestic bathtub, may be encountered as a further variant of ‘battered child syndrome’ [5,17,18]. Our own retrospective analysis of the fatalities investigated in the Institute of Forensic Medicine, University of Cologne, in the period 1980–1993 revealed 215 fatalities in the bath involving 12 children (7 boys and 5 girls), aged from 9 months to 13 years [19]. (A survey of the essential findings is given in Table 20.2.) As to the manner of death, 7 fatalities were determined as accidents, 2 as natural deaths, 1 as homicide, and 2 remained undetermined. The causes of death were drowning (5), scalding burns (2), seizure, Hurler’s syndrome and stabbing. In one of the undetermined cases, death was probably due to seizure, subsequent fall and drowning. In the remaining case the pattern of injury encountered at autopsy was indicative of a ‘battered child syndrome’ and was not compatible with the parental story of a single fall in the bathtub, but the causal relationship between maltreatment and the lethal subdural haematoma could not be established with the certainty required for legal purposes.

More detailed analysis of the age distribution shows that the deceased children >4 years old make up a non-homogeneous population comprising the natural deaths, the homicide and the undetermined cases. In contrast, the children aged from 9 months to 30 months have without exception died due to accidental drowning or scalding. In 3 cases the children had been left unattended in the bathtub for a ‘short time’ (2–15 minutes), and in 2 cases they had been left in the care of an older sibling. The depth of the water was ≤16 cm, with the surface of the water not exceeding the level of the navel.

The following brief reports of typical cases illustrate the main results of our analysis.

### Case 1

A boy, aged 13 months, was left unattended sitting and playing in a bathtub filled with water up to the level of his navel. Returning 2 minutes later, his mother found him face down in the water. The autopsy revealed ballooning of the lungs and aspiration of gastric content. There were no external injuries and he had no pre-existing diseases.

### Case 2

A girl, aged 19 months, was left in the bathtub in the care of her 3-year-old brother. The older sibling turned on the hot water tap, and the girl sustained a lethal scalding burn of approximately 90 per cent of her body surface, leaving only her head undamaged.

### Case 3

After having taken a bath with her 18-month–old daughter, a mother left the bathtub to dry her hair while the infant

**Table 20.1 Death of children in the bathtub: Survey of the literature**

| Reference | Material | *n* total | *n* child | Manner of death | Cause of death | Circumstances |
|---|---|---|---|---|---|---|
| Birrel and Birrel [5] | Melbourne, 8 case reports, maltreatment | | | | | 1 grossly ill–treated, left drowning by mother, rescued by sibling |
| Budnick [6] | USA, 1979–1982, electrocution | 95 | Highest mortality | | Electrocution | |
| Budnick and Ross [7] | USA, 1979–1981, drowning | 710 | 182 | | Drowning | Risk indicator: lack of supervision |
| Carnier [8] | Case report | | 2 | Accident | Electrocution | ♂ 3 years, ♂ 7 years, left alone for several hours; bathroom heated by portable electrical heater |
| Devos et al. [2] | Ghent, 1934–1983 | 36 | 9 | 1 homicide<br>2 accidents | Drowning<br>Drowning<br>6 CO intoxications | ♀ 5 years, ♀ 15 months, left unattended |
| Gilg [9] | München, 1964–1987, electrocution | 93,<br>16 accidents | 2 | Accident | Electrocution | Mother tried to hold slipping child, hair dryer fell into water |
| Krauland [10] | Berlin, 10–year period | 26 | 1 | Accident | Drowning | ♀ 7 years, unattended with sibling who assumed that she had lain down to sleep |
| Krüger et al. [4] | Rostock, 1969–1985 | 46 | 3 | Homicide | 1 by drowning<br>2 by stabbing | ♀ 23 months, preceding manual strangulation<br>♂ 5.5 years, ♂ 11 months siblings stabbed by mother (schizophrenia) |
| Mätzler [11] | Case reports | | 3 | 2 accidents | 1 by electrocution<br>1 by drowning | ♂ 13 years, radio fallen into bathtub<br>♂ 3.5 years, left unattended for a few minutes, secondary drowning |
| Pearn and Nixon, 1977 [13] | Brisbane, 1971–1975, bathtub immersion | | 8 | Accident | Drowning | ♀ 5 years |
| Pearn et al. [12] | Honolulu, 1973–1977, bathtub immersion | | 2 | Accident | Drowning | |
| Püschel et al. [14] | Hamburg, 1971–1983, electrocution | 49 | 3 | Accident | Electrocution | 2 years, 2 years, ♀ 13 years |
| Schneider [15] | Berlin, 1974–1984, electrocution | 49 | 4 | Accident | Electrocution | Aged 2–7 years |
| Sturner et al. [16] | Dallas, 1970–1973 accidental asphyxia deaths of children | | 7 | Accident | Drowning | 4 ♀, 3 ♂,<br>7 months to 2 years,<br>4 tub-sharing with brother,<br>3 left alone |
| Trübner and Püschel [3] | Hamburg, 1971–1988 | 245 | 24 | 20 accidents<br>2 homicides<br>1 natural death<br>1 undetermined | 8 by CO intoxication<br>5 by electrocution3 by scalding<br>3 by drowning<br>1 by autoerotic death<br>1 by drowning<br>1 by strangulation<br>Seizure | Subsequent drowning<br>Maltreatment |

*Source:* From Schmidt P, Madea B. *Forensic Sci Int* 1995;**72**:147–155.

**Table 20.2 Deaths in the bathtub involving children: Essential findings of the Institute of Forensic Medicine, University of Cologne, 1980–1993**

| Sex | Age | Manner of death | Cause of death | Water | Submersion | Signs of drowning | Injuries | Circumstances |
|---|---|---|---|---|---|---|---|---|
| ♂ | 13 months | Accident | Drowning | To navel | ? | Yes | No | Left unattended for 2 min |
| ♀ | 18 months | Accident | Drowning | Yes | ? | Yes | No | Mother erroneously afraid of electrocution |
| ♀ | 9 months | Accident | Drowning | To navel | Head | (Yes) resuscitation | No | In the care of the 2-year-old brother for ~ 5 min |
| ♂ | 13 months | Accident | Drowning | 10 cm | No | ? | No | Left unsupervised for a 'short time' |
| ♀ | 30 months | Accident | Drowning | 8 cm | Head | Yes | Small contusion of the scalp | Left alone for ~ 15 min |
| ♀ | 19 months | Accident | Scalding | Yes | | No | Scalding burns involving 90% of body surface | Left unattended with 3-year-old brother who turned on hot water |
| ♂ | 14 months | Accident | Scalding | Yes | | No | Scalding burns | Put by the mother into hot water which had been let in by an older sibling |
| ♂ | 13 years | Natural death | Seizure | Yes | Head | No | No | Known history of epilepsy |
| ♂ | 9 years | Natural death | Hurler's syndrome | Yes | ? | No | Discrete scalding burns | Known history of Hurler's syndrome, mentally incapacitated |
| ♀ | 13 years | Homicide | Stabbing | No | No | No | 17 stab wounds of chest and abdomen, gag, tying | Deposition of the corpse in the bathtub |
| ♂ | 4 years | Undetermined | Subdural haematoma | | | | Multiple bruises of different ages at head, trunk and extremities, fracture of a finger | Mentally retarded, discrepancy between the parental story of a single fall in the bathtub and the pattern of injury, maltreatment |
| ♂ | 10 years | Undetermined | ? | 16 cm | Head | ? | External examination: contusion of the forehead and abrasion of the skin | Known history of epilepsy, assumption of fall due to seizure and subsequent drowning |

*Source:* From Schmidt P, Madea B. *Forensic Sci Int* 1995;**72**:147–155.

remained playing in the water. The girl suddenly tried to stand up, slipped and plunged into the water. When the mother tried to help her, she slipped on the wet floor and the hair dryer fell into the bathtub. Afraid of electrocution, she ran out of the bathroom to interrupt the electrical supply and call the emergency doctor. On her way back she collapsed. The emergency doctor removed the child from the bathtub without being electrocuted. The autopsy did not reveal electrical burns but ballooning of the lungs indicating lethal drowning.

## Case 4

According to the statements of the mother and her friend, the 4-year-old boy who was mentally retarded had slipped in the bathtub and fallen on his head. One hour later he began to lose consciousness so the mother called the emergency doctor who immediately admitted the boy to hospital. Computer tomography of the head showed a large subdural haematoma covering the right hemisphere of the brain, general cerebral oedema and displacement of the right hemisphere. Despite emergency surgical removal of the haematoma, the boy died 3 days later. The autopsy revealed an extensive contusion of the scalp at the left side of the skull, older cutaneous haemorrhages of the right temple, the left cheek and the chin, as well as multiple bruises of different ages widely distributed over the extremities and the trunk including the buttocks, and, furthermore, a fracture of the second finger of the right hand. Neuropathological examination showed residues of a subdural haematoma over the right hemisphere, contusions at the base of the left frontal lobe and severe hypoxic neuronal changes. The pattern of these lethal injuries could only partly be related to a single fall. The multiple bruises and haemorrhages of the face and extremities could not be explained by the parental story. To sum up, the findings indicated a 'battered child syndrome' but a causal relationship between the repeated maltreatment and the lethal outcome could not be proved with sufficient certainty.

## ■ Discussion

Considering the age distribution, Pearn and Nixon [13] supposed that two separate populations might account for bathtub immersion. This conclusion is confirmed by the analysis of our series. The deceased children >4 years old make up a heterogeneous group accounting for the natural deaths, the homicide and the undetermined cases while the younger victims without exception died due to accidents, drowning or scalding. The occurrence of two fatalities probably owing to seizure is in good agreement with the majority of studies on death in the bathtub identifying epilepsy as one of the more important causes of natural death [2,3,11,20]. Analysis of 710 bathtub-related drownings [7] pointed out that a history of seizure disorder was the most common risk indicator reported among persons aged 5–39 years. The fatality due to subdural haematoma additionally showing the characteristic signs of maltreatment [9,19] is in keeping with the evidence provided by Nixon and Pearn [17,18] that deliberate immersion of a child under water, particularly in the domestic bathtub, may be encountered as a further variant of battered child syndrome. As attempted intentional drowning does not leave pathognomonic stigmata, the authors have pointed out the common characteristics of non-accidental immersion: child aged 15–30 months, may be handicapped, often the eldest child of a small sibship, alone in the bath at an unusual time of the day, parents with full sociopathology of inflicting non-accidental injury and acute parental stress, often domestic altercations, as precipitating cause.

Considering the scene of the toddlers' accidental drowning the question arises as to what amount of water is required to cause the fatal outcome. Results of animal experiments and clinical observations [21–23] indicate that inhalation of >20 ml fresh water per kg of body weight within a few minutes may produce severe persisting electrolyte changes (hyponatraemia, hypochloraemia and hyperkalaemia) which may contribute to ventricular fibrillation. Aspiration of 11 ml/kg fresh water caused an increase in blood volume in direct proportion to the quantity of fluid aspirated. Inhalation of 1–3 ml/kg was followed by pulmonary hypertension due to arteriolar vasoconstriction and airway closure due to contraction of the musculature of the terminal bronchioles resulting in 75 per cent of blood perfusing non-ventilated areas [24–28]. These changes are mediated as a local parasympathetic reflex [24,28] and are appropriate to cause hypoxaemia, acidosis and lethal cerebral hypoxia [21,22,27,28]. This condition, known as 'dry drowning' or probably more correctly 'death from submersion', is estimated to account for 10–20 per cent of fatal drowning [21,27,28]. These findings explain that a lethal outcome may occur even at the depth of the water encountered in our material. This is in keeping with the results of Australian investigators [13] who in a review of 19 bathtub immersion accidents involving children found a median depth of the water of 20.3 cm with the range being 5.1–35.6 cm.

In this context the observation that some children who lost their balance and fell into shallow water got into a state of shock, immediately lying face down in the water without struggling, is of interest. (Krauland [10] reported that a 6-year-old boy who observed his sister drowning in the bathtub thought that she had lain down to sleep. Diagnosis of drowning was confirmed and internal disease excluded by autopsy.) This behaviour may support the development of a rapid and efficient diving reflex [29] but may gain fatal relevance if the children are not rescued before they start to aspirate water or hypoxic damage occurs to the brain.

A further probable question may be whether the short time of lacking supervision reported by the parents is sufficient to cause lethal drowning. In the study cited earlier [13], the immersion time ranged from 3–5 minutes with a median value of 4 minutes in survivals and 3–20 minutes with a median value of 5 minutes in fatalities. The authors drew the following conclusions. Consciousness is lost within 3 minutes of involuntary submersion due to cerebral hypoxia. Children immersed for less than 3 minutes are likely to survive. The estimates of immersion time in warm water for children's fatalities are in the range of 3–10 minutes. As a consequence, the difference between a survival and a potential fatality may depend on differences in immersion time of around only 1 minute [13,28]. Being left unattended was emphasized as the major personal risk indicator in bathtub–related drownings among children <5 years old [7,12].

The findings of the present study support the recommendations proposed by these authors: No child of less than 3 years of age should be left in the bath without surveillance because of the risk of drowning, and no child less than 7 years old should be left unsupervised due to the danger of inadvertent hot water scalds [7,12].

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# 21

# Obstruction of the Respiratory Orifices, Larynx, Trachea and Bronchia

*Wolfgang Keil*

## 21.1 Obstruction of the mouth and nose

### Definition

This typifies asphyxiation due to constrictions or obstructions of the mouth and nose, also termed ‘oronasal occlusions’. In English literature, the term ‘smothering’ has often been used for this type of asphyxiation [1,2].

### Forms

Three groups may be distinguished.

#### Manual obstruction of the respiratory orifices

In some cases a perpetrator applies minimal pressure to cover the mouth and nose of his defenceless victim. Alternatively, the perpetrator may use violent force to squeeze the mouth and nose shut with his hands. Forcible closure of the respiratory orifices can also be caused by pressing the victim’s face against the assailant’s body.

#### Obstruction of the respiratory orifices by means of objects

This includes asphyxiation using a soft covering. In this case, soft objects such as pillows and plastic sheets slip over the mouth and nose or are placed over them. This form of asphyxiation also includes using such soft objects to hold the respiratory orifices closed or cover the face [3]. This category also covers the binding, sealing and occlusion of the mouth and nose, the latter including gagging. Gags, predominantly made of cloth, are stuffed into the oral cavity, sometimes as far as the pharynx, and secured with a tape running through the mouth. Frequently restrained using shackles, the victim then breathes mainly through the nose. If the gag is pressed far back in the throat, contractions of the pharyngeal muscles may initially keep an airway open for nasal respiration. Asphyxiation is possible when the muscles slacken.

#### Obstruction of the respiratory orifices by facial occlusion

Individual fatalities are evidently possible due to pressing or lying on the face without external intervention. As a general rule, this affects persons who are debilitated due to illness or intoxication [4]. In homicidal asphyxiation, the perpetrator presses the victim’s face into soft objects such as bed linen.

### Frequency/occurrence

To our knowledge, such fatalities are encountered relatively rarely in forensic pathology practice. However, it should be borne in mind that oronasal occlusions may not exhibit any injuries. In view of the unspecific general findings, it is inevitable that such cases of asphyxiation are not discovered [5]. It is not possible in practice to distinguish these cases from sudden infant death syndrome (SIDS) due to the findings [6].

It may be assumed that most victims of asphyxiation by means of soft coverings are newborns and infants. As evidenced by individual case histories, the frequency of such homicides with adult or elderly victims is estimated to be considerably lower. The physical superiority of the perpetrator is a contributory factor in the event of foul play. As a result of illness, infirmity or intoxication, adult victims may have limited or suppressed ability to act and resist. In isolated cases, victims are restrained by one or more perpetrators while a further assailant presses a pillow over their mouth and nose.

Cases of asphyxiation caused by binding, sealing or obstructing the respiratory orifices, or gagging, are extremely rare. Gagging is often found in conjunction with other traumatic violence, which is the actual cause of death. If applicable, it must be evaluated whether the gag was a concurrent cause of death. If the passages of the mouth and nose are still partially open or where complete obstruction of the airways was only short-term, the $O_2$ deficiency may constitute a contributory factor for the onset of death. In exceptional cases, the obstruction of the respiratory orifices merely represents a secondary finding.

Rarely encountered, accidents are mostly caused by bed linen or pillows. The polythene sheets used in plastic shopping bags or as packaging for magazines may occasionally cause fatal accidents [3].

### Classification of the circumstances

Since the mechanisms in asphyxiation by means of soft coverings are mostly pillows, covers and scarves, and more rarely items of clothing, this type of object may be present when the body is discovered. Numerous victims are apparently found in bed, particularly infants.

## Pathomorphology

### External findings

**Petechiae** Individual petechiae may develop in the conjunctivae, very seldom in the oral mucosa and the skin of the face.

**Facial injuries** Abrasions may be found (Figure 21.1). Small haematomas are also observed. The findings may not only be located in the vicinity of the mouth and nose but also in other regions [7]. There may be wounds in the lip vermilion and the oral mucosa; this finding is most common in adults.

**Residue of adhesive material** The adhesive surfaces of plasters or duct tape can leave a residue, which must be retained. Where the material has since been removed, sharply delineated desiccations may develop postmortem in the skin of the face, corresponding to the location of the original adhesive zone.

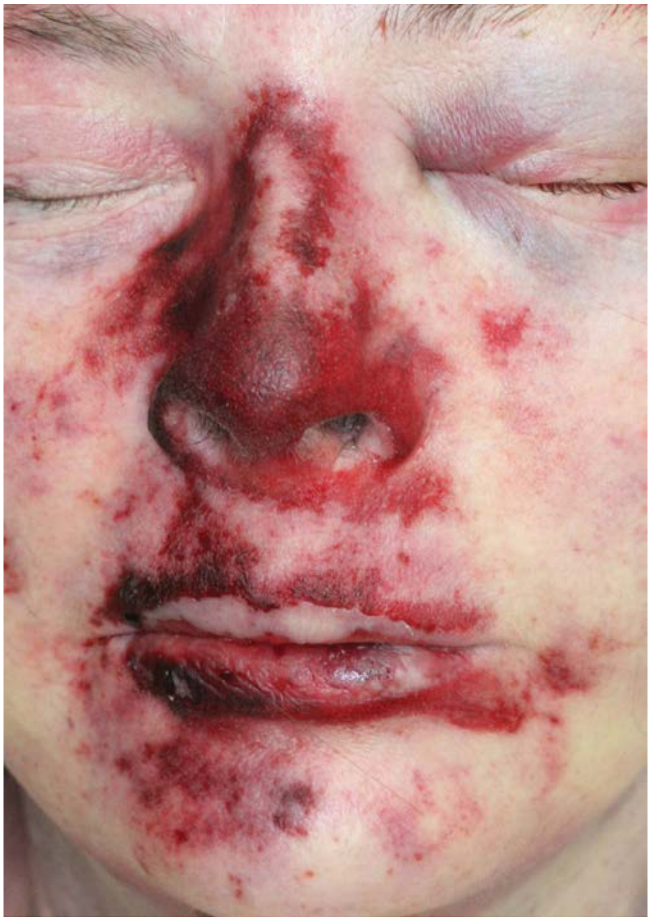

**Figure 21.1** Obstruction of the mouth and nose – apparent homicide by the husband. A towel with a rough surface was held over the respiratory orifices. Abrasions around the nose, the lip vermilion and above the chin.

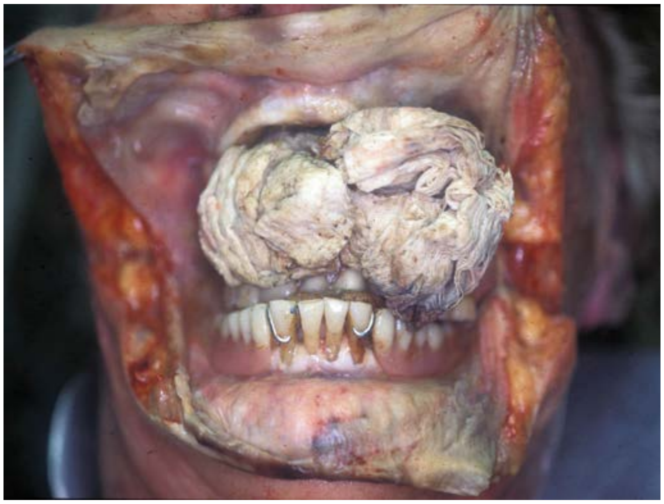

**Figure 21.2** Dissection technique where the gag is still present – transversal incisions extending from both corners of the mouth.

**Foreign matter in the respiratory orifices** Any textile fibres that could be attributed to an asphyxiation instrument and any other residues must be preserved.

**Dissection where gag is still in place** If a gag is still present, this is left in place at the beginning of the autopsy. First of all, a description of any facial injuries is prepared. Hereafter, incisions are made from the corners of the mouth extending transversally over both cheeks, exposing the oral cavity and nasopharynx, in order to ascertain the extent of the gagging. This procedure serves as the basis in determining whether the gag can be considered the cause of death. A photographic documentation should record the various phases of the dissection (Figure 21.2).

**Foam on the mouth and nose** A fine, white foam, occasionally streaked with blood forms.

**Injuries in other regions of the body** Abrasions, haematomas and wounds may be present. Particularly in the case of infants and small children, any indications of previous as well as recent traumatic violence should be noted. Any restraint or defence injuries and marks of shackles must be taken into consideration.

### Internal findings

**Petechiae** Minimal petechiae may be established under the pleura pulmonalis and under the fasciae of the musculi temporales, or not at all. In rare cases, they may occur under the epicardium, in infants and children also under the thymus capsule.

**Formation of foam in the airways** Occasionally, a fine, white foam may adhere to the upper and lower respiratory walls, sections of which may be streaked with blood. Excessive mucus may be present.

**Acute pulmonary emphysema** The finding may affect both lobes or only individual sections of the lung.

**General findings** Usually liquid blood and acute hyperaemia of the internal organs.

## Differentiation between suicide, homicide, accident and natural death

### Suicide

In exceptional cases, suicide has been reported by means of self-inflicted obstruction of the mouth and nose.

### Homicide

As a rule, the forensic medical findings only suffice to indicate the eventuality of external intervention (e.g. due to injuries to the face, nose and mouth). Additional injuries in other regions of the body may substantiate the suspicion of foul play.

### Accident

Depending on the anamnesis and the circumstances of the demise, the possibility of an accident cannot be ultimately ruled out where injuries are only minimal or non-existent.

### Natural death

Cause of death may be due to pre-existing conditions or acute symptoms of disease. During the process of dying, decedents may adopt a facial position, which, depending on the circumstances, is to be considered an acceleratory factor for the onset of death but not the underlying cause.

# ■ Obstruction of the larynx (bolus death)

## Definition

Foreign material which completely fills or tampers out the larynx also stops the air supply at the same time. If death suddenly occurs, it is nonetheless not a form of asphyxiation, but a reflex death with sudden cardiac arrest, called bolus death (see Chapter 26).

# ■ Obstruction of the trachea and bronchia

## Definition

Asphyxiation due to obstruction of the trachea and/or bronchia is caused by aspiration of material originating in the body or a foreign body.

Aspiration is not only a possible cause of death but also of great relevance as a vital sign.

The term ‘choking’ should no longer be used for obstructions of the trachea and bronchi due to the inconsistent usage for other forms of asphyxiation (see Chapter 1, Table 1.8).

## Forms

### Aspiration of material originating in the body

A particularly significant role is played by the aspiration of chyme and blood. Only in cases where at least the small bronchi on both sides are obstructed by the aspirate up to the periphery is the evaluation of aspiration of chyme or blood as the cause of death justified. Some of these cases are accompanied by the development of areas of chyme and blood aspiration. An acute pulmonary emphysema may frequently be observed. If aspirated material is only found in the trachea and the primary bronchia, and this section of the airways is not completely obstructed, death by asphyxiation may not be assumed. These findings may be interpreted as agonal aspiration with another underlying cause of death. In rare cases, teeth are aspirated; although this may result in respiratory impairment, this generally does not cause death by asphyxiation.

### Aspiration of foreign bodies

Aspirates may be peanuts, small sweets, fruit stones, parts of toys, metal objects such as screws and nails, or parts of dentures. Grains are aspirated when victims are engulfed in grain elevators. Rare cases of aspiration of sand and soil have been documented. Fatal foreign body aspiration occurs primarily in cases involving infants and small children. Due to the much larger diameter of the trachea and bronchia, most older children and adults only have difficulty breathing. In rare cases, aspiration of amniotic fluid causes death by asphyxiation in newborns. Likewise, cases of drowning may be classified in this category; the diagnosis ‘death by drowning’ signifies that asphyxiation was caused by inhaling the drowning liquid, which is water in virtually every case.

## Frequency/occurrence

Vomiting may be caused by numerous severe internal diseases, injuries and intoxications. Aspiration of chyme or gastric contents occurs especially where individuals are in a reduced state of consciousness or even unconscious, thus their protective reflexes are suspended [8]. As these circumstances are not particularly unusual, chyme aspiration is the most common form of aspiration. Especially agonal inspiration may be frequently encountered. Fatal chyme aspiration is predominantly found in cases of intoxication, particularly those induced by heroin, and in craniocerebral trauma. Gastrointestinal diseases such as ileus frequently induce vomiting and may ultimately cause fatal aspiration of the stomach contents. The eventuality of chyme aspiration should always be taken into consideration in diseases featuring dysphagia. Patients in care facilities are predominantly affected. Asphyxiation due to aspiration of blood is a regular occurrence in forensic pathological routine work. Sudden natural deaths here primarily include oesophageal variceal haemorrhages, which, following substantial haemoptysis, ultimately lead to aspiration of blood and asphyxiation. However, the source of the haemorrhage may also be located in the lungs, as in the extremely rare condition of Wegener's granulomatosis. Moreover, patients who are prescribed anticoagulants following tonsillectomies and tooth extractions may develop complications, one of which is the aspiration of blood. Paramount traumatic causes include craniofacial and subcranial fractures, as well as pulmonary contusions, ruptures and lacerations.

By comparison, aspiration of foreign bodies is rare, occurring most commonly in older infants and small children, who generally tend to put objects in their mouths. Occasionally, adults aspirate nails or screws held between their lips while carrying out manual jobs. In old people's homes and care facilities, it is important to bear in mind that teeth or parts of dentures that fall out may have been aspirated unnoticed. In exceptional cases, such aspirates cause fatal inflammations of the airways and lungs.

## Pathomorphology

### External findings

**Vomitus or blood on the face** In chyme aspiration, vomitus is frequently found on the face, predominantly around the mouth and nose. In blood aspiration, there may be tracks where blood has flowed from the nose and mouth. Evidence of haemoptysis may be visible in other regions of the body (e.g. on the hands).

**Aspirate in mouth and nose** Vomitus or blood may be found around the mouth and/or nose.

**Facial injuries** In view of possible craniofacial fractures and other traumatic haemorrhage sources, it is mandatory to identify any injuries to the skin, particularly in the face.

**Location of haemorrhage sources in the nose, mouth and throat** The inspection may yield evidence of any haemorrhage sources in these regions. Overlying blood clots may be crucial evidence of this.

**Petechiae** Particularly in extensive or complete obstruction of the airways, there may be minimal petechiae in the conjunctivae, or none at all.

### Internal findings

**Aspirate in the airways** Asphyxiation is evidenced by the presence of the aspirate (Figure 21.3). It should be noted, however, that parts of the aspirate at least may have been removed in the course of resuscitation measures.

**Aspiration areas in the lungs** These occur in cases of chyme and blood aspiration (Figures 21.4 and 21.5). If large quantities of acidic gastric juices are aspirated when vomiting chyme, brownish grey areas of preliminary digestion may form on the tissue of the lungs (Mendelson's

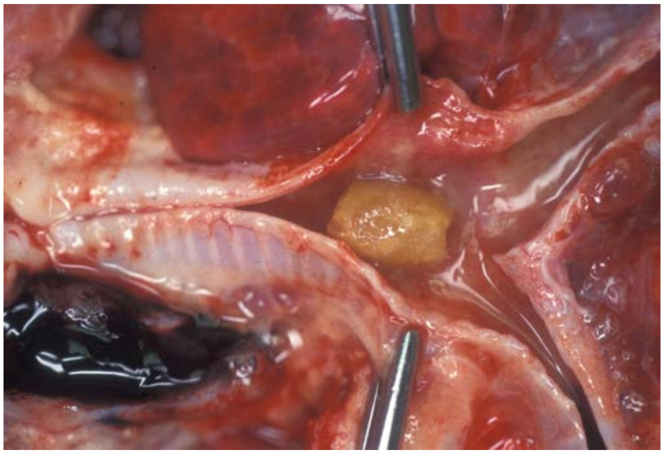

**Figure 21.3** Foreign body aspiration – obstruction of the trachea of an infant by a peanut.

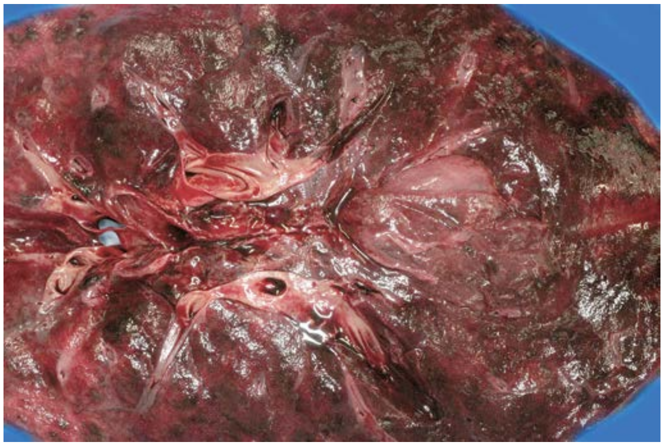

**Figure 21.4** Chyme aspiration areas in the case of heroin intoxication. Areas exhibit greenish-brown discolouration.

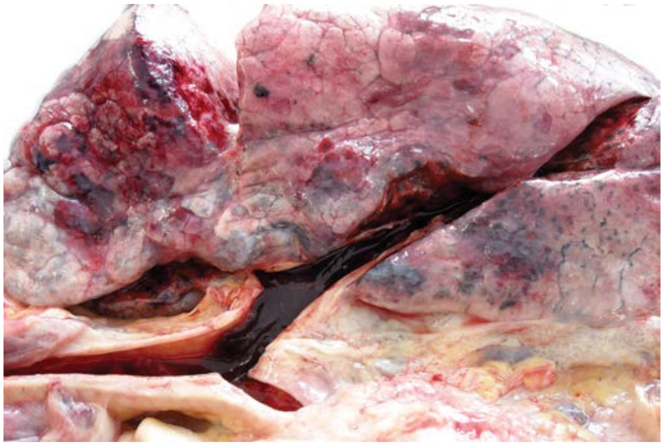

**Figure 21.5** Aspiration of blood following a craniofacial fracture. Blood is present in the right primary bronchus, blood aspiration areas under the pleura pulmonalis and on the pulmonary section, and acute emphysema of the anaemic lung.

syndrome). Furthermore, many cases exhibit a pronounced pulmonary oedema. Where this does not prove fatal, aspiration pneumonia subsequently develops. Postmortem CT imaging may be helpful in assessing the severity of aspiration [9,10].

**Acute pulmonary emphysema** This finding is frequently observed. As a rule, the emphysema is particularly intensive in cases where the obstruction of the airways was caused by mainly liquid material. The pulmonary surface then displays suspended retraction, like that observed in drowning.

**Content of the oesophagus** Large quantities of chyme, first vomited then aspirated, are commonly found in the oesophagus. In blood aspiration, blood may be present.

**Haemorrhage source** In cirrhosis of the liver, particular attention should be paid to any ruptured oesophageal varices. On occasion, a gastric ulcer may be the source of the haemorrhage. If there is reason to suspect that lung tissue is the source of the haemorrhage, additional histological and serological examinations will be necessary where no relevant syndrome was diagnosed antemortem. Any skull fractures, injuries to the soft tissues in the head and neck region and lung injuries that could be the origin of blood aspiration must be established.

**Petechiae** There may be minimal petechiae on the serous membranes or none at all.

**General findings** Fluid blood in the heart and acute blood load of the internal organs. Considerable brain oedema occurs.

### Differentiation between suicide, homicide, accident and natural death

#### Suicide

Chyme aspiration occurs primarily in suicidal intoxication due to heroin but also in the course of intoxication caused by various tablets. Fatal blood aspiration is also observed in suicidal cut throat injuries. Intensive blood aspiration is occasionally diagnosed in suicidal decapitations by train impact. In such cases, the blood aspiration is significant, not as the cause of death but as a vital sign.

#### Homicide

Complex blunt force trauma, particularly craniocerebral trauma, may ultimately result in vomiting with fatal chyme aspiration. If persons suffering from dysphagia are fed too hurriedly, causing fatal aspiration of chyme, it could be argued that this constitutes involuntary manslaughter. Blood aspiration may also originate from craniofacial fractures or injuries to the lungs. Stab wounds or incisions to the throat that cut open the arteriae carotides and the trachea or the larynx at the same time may lead to severe blood aspiration, which frequently constitutes the cause of death in conjunction with the loss of blood.

#### Accident

This generally applies to craniocerebral trauma accompanied by vomiting and possible aspiration of chyme. By comparison, fatal blood aspiration is less relevant to the onset of death in accidents. Asphyxiation due to foreign body aspiration in infants and small children may be considered accidental.

## 21.2 Oxygen deficiency in the tidal air

■ *Wolfgang Keil*

This type of asphyxiation is often referred to as ‘suffocation’. However, this term is not specific and has been used for various types of asphyxiation (see Section 2, Table 6.1). Therefore, the term should no longer be used in forensic pathology.

### ■ Normobaric $O_2$ deficiency

#### Definition

This refers to asphyxiation due to the reduction of the $O_2$ concentration in the tidal air at constant air pressure.

#### Forms

##### Reduction of the $O_2$ concentration by the expired air

This constellation presupposes an enclosed volume of air available for respiration. A self-contained volume exists where individuals are imprisoned in sealed containers, virtually airtight pieces of furniture or boxes. This special form of normobaric-$O_2$ deficiency is occasionally referred to as entrapment in English forensic pathology. In the same way, placing a plastic bag over the head also results in a virtually enclosed volume of air. The limited volume leads to the repeated inhalation of expired air, a process also known as rebreathing. As the $CO_2$ content equals approximately 0.04 per cent by volume in the atmospheric air and approximately 4.04 per cent by volume in the expiration, the proportion in the inhaled air increases rapidly. At the same time, the $O_2$ content in the enclosed air volume decreases constantly until it reaches the lethal concentration of approximately 10 per cent by volume. The heightened $CO_2$ content in the inhaled air leads to an increase in the respiratory rate, accelerating the asphyxiation process. A similar pathomechanism occurs in diving in the event of a malfunction in closed-circuit breathing apparatus.

### Decrease in $O_2$ concentration by an influx of gases

Possible causes may be industrial accidents in which there is a leakage of technical gases, thereby decreasing the $O_2$ concentration of the atmospheric air. The formation of large quantities of $CO_2$ induces an $O_2$ deficiency near sources of fire. The gases that accumulate in or are fed into silos, wine cellars and tanks can significantly reduce the $O_2$ content, leading to rapid asphyxiation in individual cases. It should be noted that not only does the influx of gas decrease the $O_2$ content of the air, but the gases themselves also have toxic effects. A $CO_2$ concentration of 8 per cent by volume and above is potentially lethal. In operating theatres or emergency health care, exceptional cases may occur in which $O_2$ gas cylinders are inadvertently swapped with cylinders containing other gases, which could cause asphyxiation.

## Frequency/occurrence

According to studies in Berlin and Vienna [11,12], cases of plastic bag asphyxiation occur with a frequency of less than 0.3 per cent in autopsy material. Fatalities due to other types of normobaric hypoxia are extreme exceptions. Right-to-die organizations propagate plastic bag asphyxiation as a method of suicide. For some years now, so-called exit bags filled with helium have also been used, which shortens the duration of the asphyxiation process [13]. This form of death may also be observed in cases of autoerotic activity. On average, suicides are a great deal older than individuals who may be involved in sexual practices. Occasional deaths occur when individuals sniff specific substances inside the plastic bag to enhance stimulation or intoxication [14].

## Classification of the circumstances

In virtually every case, the facts regarding the circumstances of the death are essential for the overall evaluation [15]. The circumstances are also relevant for the subsequent reconstruction of events. However, since changes are made to the scene of death in most cases for a variety of reasons, evaluation may prove difficult. Where suicide notes and personal documents such as testaments are laid out, this may constitute evidence of a suicide. Occasionally, the sexual nature of the circumstances is apparent.

## Pathomorphology

### External findings

**Plastic bag fatalities** It must be established whether the bag is tied around the neck (Figure 21.6). However, asphyxiation may still occur if the bag is placed over the head and left open at the bottom. The bag must be preserved in order to carry out chemical–toxicological screening for substances on the inner surfaces. Condensation on the inside of the bag does not prove that exhalation took place in the bag as such condensation may be produced postmortem [16].

**Petechiae** These are rarely observed in the conjunctivae and the skin of the eyelids, and there may be none.

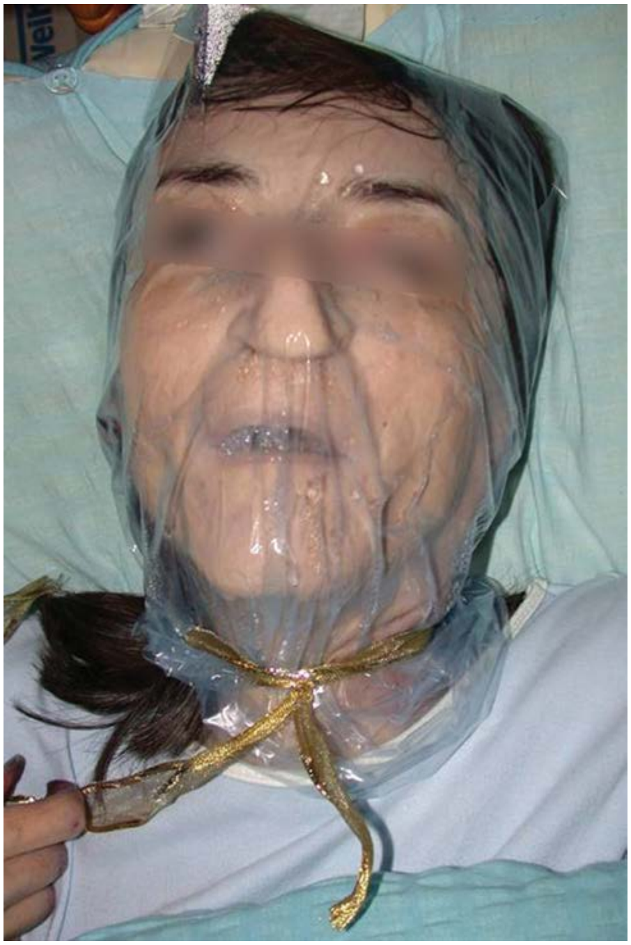

**Figure 21.6** Rebreathing – suicide by use of plastic bag. No relevant anatomical pathological findings.

**Injuries** Injuries in all regions of the body, particularly evidence of a struggle or defence injuries, must be taken into consideration.

### Internal findings

**General findings** Liquid blood and acute hyperaemia of the internal organs. Congestion above the level of the clavicles is observed. Petechiae in the conjunctiva and under the pleura may occur. Inhalation of helium may also cause pulmonary oedema. Brain oedema.

### Additional examinations

**Histological examinations** These should always be carried out in order to eliminate any pre-existing physical conditions.

**Blood alcohol test** With regard to the overall evaluation, this test must be carried out.

**Chemical–toxicological analyses** These analyses are mandatory, both for the cadaver material and for any contents of the bag [17].

**Measurement of the gas concentration at the scene of death** Such measurements must be undertaken by specialists in order to corroborate the diagnosis of asphyxiation or, for example, to consider a possible $CO_2$ intoxication.

### Differentiation between suicide, homicide, accident and natural death

#### Suicide

Of the asphyxiation mechanisms mentioned, suicide by plastic bag asphyxiation would appear to be the most common. However, the forensic medical findings may only suffice to rule out any other traumatic violence, especially evidence of a struggle. When using this method, suicides occasionally take sedatives and antiemetics. In particular, the detection of antiemetics may support the hypothesis of suicide.

#### Homicide

Isolated homicides have been documented in which plastic bags were placed over the victim's head [14]. Perpetrators have been known to kill their victims using another form of traumatic violence and then place a plastic bag over their heads in the final stages of the homicide. The medical findings in such cases cannot assist in clarifying whether the plastic bag was of any relevance for the ultimate onset of death. Homicide also includes cases of human trafficking when individuals die of asphyxiation while being transported in containers. Cases of homicide have been recorded in which victims are shut in cupboards or boxes, which are then buried.

#### Accident

Accidents with plastic bags occur in association with autoerotic activities. The sexual nature is indicated only by the circumstances at the scene of death. Accidents may happen without any sexual aspect where individuals sniff solvents, for example, from plastic bags to attain a state of intoxication. In rare cases, small children asphyxiate after pulling a plastic bag over their own heads. It is not possible to differentiate between an accident, suicide or homicide in every case of plastic bag asphyxiation. Deaths in silos, accessible tanks and airtight pieces of furniture almost invariably constitute accidental asphyxiation. The same applies to the leakage of technical gases in industrial incidents.

#### Natural death

Occasionally, $O_2$ deficiency may bring about deaths by natural causes, particularly where there are pre-existing physical conditions. For example, these conditions may lead to cardiac deaths or fatal status epilepticus.

## ■ Hypobaric $O_2$ deficiency

### Definition

This refers to asphyxiation due to the reduction of partial $O_2$ pressure in the tidal air at constant $O_2$ concentration.

### Frequency/occurrence

This type of asphyxiation is very rarely observed. Fatalities only occur at high altitudes, for example while mountaineering, in cable cars, hot air balloons or aeroplanes. The decreasing partial $O_2$ pressure is responsible for altitude sickness. This involves hypoxic pulmonary vasoconstriction, whereby the lungs' ability to absorb $O_2$ is diminished. Moreover, a pulmonary oedema develops due to hypertension. The body's respiratory control does not counteract the mechanism as it responds primarily to the $CO_2$ content of the blood, which, however, does not increase due to decreasing air pressure.

Isolated fatalities have been reported, which may apparently be attributed to a failure of the pressure control system in aircraft. In the event of a sudden drop in cabin pressure at an altitude of approximately 10,700 m, the passengers are only capable of self-rescue for 30–60 seconds. Incapacitation and subsequent loss of consciousness may occur within a very short time. Asphyxiation takes place immediately afterwards [18].

### Pathomorphology

#### External findings

No external findings are to be expected.

#### Internal findings

**Pulmonary oedema** This finding should be present in altitude sickness.

**General findings** The symptoms include liquid cadaver blood, acute hyperaemia of the internal organs and brain oedema.

# 21.3 Hanging

## ■ *Wolfgang Keil*

### Definition

'Strangulation' is the generic term for any form of compression of the neck. 'Hanging' is a fatal compression of the neck by means of a ligature that is constricted by the weight of the body.

## Frequency/occurrence

Throughout the world, hanging is one of the most common methods of suicide and is favoured by men. Accidents are comparatively rare [19,80] and primarily involve incidents of autoerotic asphyxiation. Near-hanging to enhance sexual stimulation is evidently almost always practised by men [2]. Accidents in which subjects fall or slip from a height and find themselves in a hanging position from which they are unable to escape by their own efforts are extremely rare [20,21]. For example, there have been cases of small children falling onto low, taut ropes resulting in death by hanging [22]. Extremely rare cases have been reported of homicidal hangings as foul play [23,24]. In some countries, hanging is still a method of execution. In a very small number of cases, the victim was killed by another method and strung up in a hanging position in order to simulate a suicide for the purpose of concealing the crime.

## Strangulation devices

For the most part, ligature materials with a circular cross section are used. The diameter varies from shoelaces to a rope. Cords made of rubber, cables and wires are also used. Ligatures that do not have a circular cross section include belts, straps or, more rarely, even chains. These types of object may produce characteristic indentations within the mark of the ligature. Occasionally, hanging is carried out using items of clothing, bed linen or towels. In exceptional cases, death by hanging may even occur on hard objects; cases of hanging have been reported in forked branches, on chair backs, hospital bed boards and on the edges of wash basins and toilets [25].

As a general rule, the conventional ligature is tied into a noose, which is passed once or twice, more rarely in multiple loops, around the neck. Where the noose is passed completely round the neck, this is referred to as a 'closed noose', while a noose with a slipknot that binds tighter when the rope is pulled is also known as a 'running noose'. An 'open noose' does not completely encircle the neck. It does not even have to be wound around the neck. The latter is found, for example, in cases of hanging on taut ropes, fixed shower hoses or bicycle helmets [26]. In these instances, the strangulation device only constricts the region located directly beneath the chin and/or the front and lateral areas of the neck.

## Classification of the circumstances

Essentially, there are two distinct situations in which the body may be found, namely incomplete and complete hanging.

### Incomplete hanging

The body is suspended but has some form of contact to the ground or the object from which the hanging took place, usually with the feet, more rarely with other parts of the body (Figure 21.7). As a result, only part of the body weight is pressing on the neck region. Therefore, this hanging position is also referred to as 'partial suspension'. Where the feet are still just touching the floor, it is common to find a climbing aid next to the body, such as a stool, used

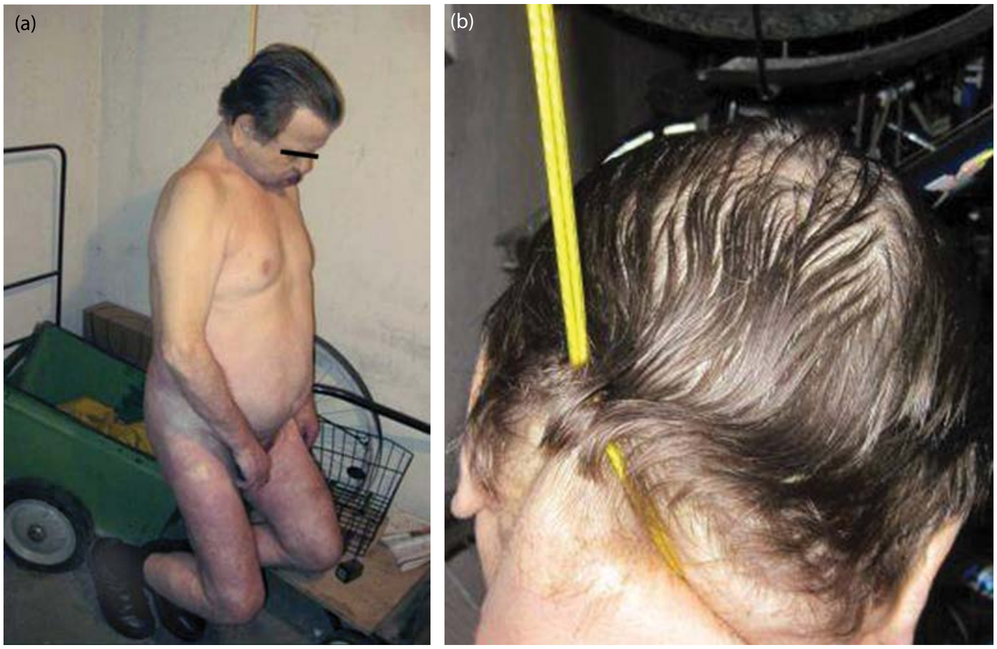

**Figure 21.7** (a),(b) Incomplete hanging – suicide. The highest point of the ligature is located on the left of the nape of the neck.

to facilitate the hanging. Occasionally, victims are also found in other positions, including sitting, crouching, kneeling or lying down. In approximately 70 per cent of all suicides by hanging, the body has direct contact of some kind with the surface below [27]. This very common position is known as 'atypical hanging' [28]. However, there is no standardized definition of the term 'atypical hanging'. Some authors describe the position as 'atypical' when the knot is not placed centrally in the nape of the neck [29]. Since the constriction of the neck by the body weight is a crucial aspect in the development of the morphological findings, the term 'incomplete hanging' would appear to be more appropriate than 'atypical hanging'.

### Complete hanging

There is no contact between the feet and the floor, i.e. full suspension is achieved (Figure 21.8). In suicide hanging, a climbing aid will always be found in the immediate vicinity of the deceased, otherwise there can be no explanation for the free suspension. In extremely rare cases, a machine may be used (e.g. a crane), with which the suicide hoists himself up to achieve full suspension. Cases of complete hanging are comparatively rare. In special publications, complete hanging is referred to as 'typical hanging'. Furthermore, individual authors stipulate that, for 'typical hanging', the ligature must be knotted centrally at the nape of the neck or at any rate behind the ears [30]. In practice it is irrelevant whether the knot is located in the middle or on the side behind the ears, since the more lateral position evidently only serves to promote the formation of petechiae [31].

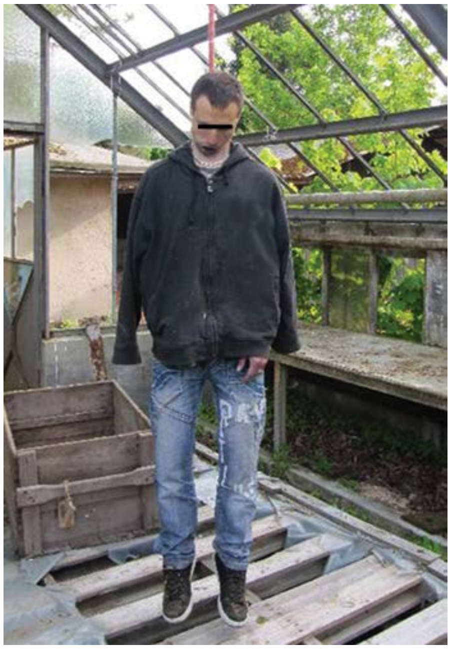

**Figure 21.8** Complete hanging – suicide. Free suspension. The highest point of the ligature is located centrally in the nape of the neck.

A distinction should be drawn in isolated cases in the 'complete hanging' category where the individuals plunge or leap from a height with the noose around their necks. A long drop is used in executions, known as judicial hangings, when the condemned prisoner falls down through a trapdoor and then hangs freely. In drops of more than 1 metre into the tightening noose, fractures of the second cervical vertebra are common in adults. There have been several reports of suicides plunging from a height with the noose around their necks. Depending on the height of the drop, the body weight and the elasticity of the ligature material, this can result in decapitation [32,33].

## Pathomorphology

### External findings

**Petechiae/congestion syndrome** Complete hanging frequently does not result in petechiae (Figure 21.9). In some cases, they may merely be present in the conjunctiva and/or in the skin of the eyelids. By contrast, in incomplete hanging, numerous petechiae are generally found spread over the entire eye area (Figure 21.10). Where the constricting force is only a small proportion of the body weight, this may lead to congestion syndrome (Figure 21.11); as a result, petechiae may also occur in the oral mucosa, in the skin of the face and behind the ears. In extreme cases, the congestion syndrome produced by incomplete hanging may resemble the appearance of a ligature strangulation [34]; thus, a differentiation is problematic at best or may even prove impossible. The petechiae are always delineated sharply from the ligature furrow downwards.

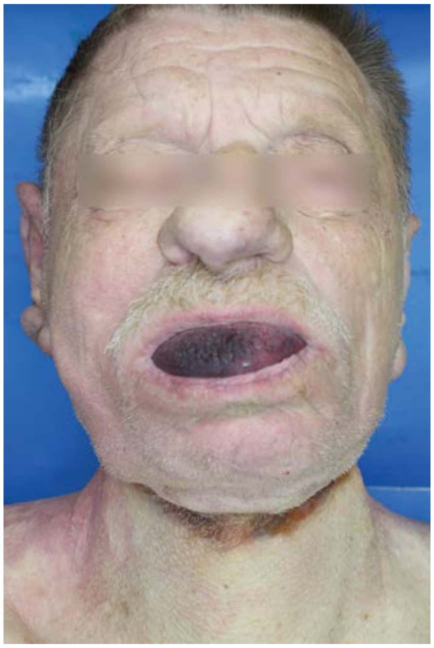

**Figure 21.9** Complete hanging – suicide. No petechiae, no congestion.

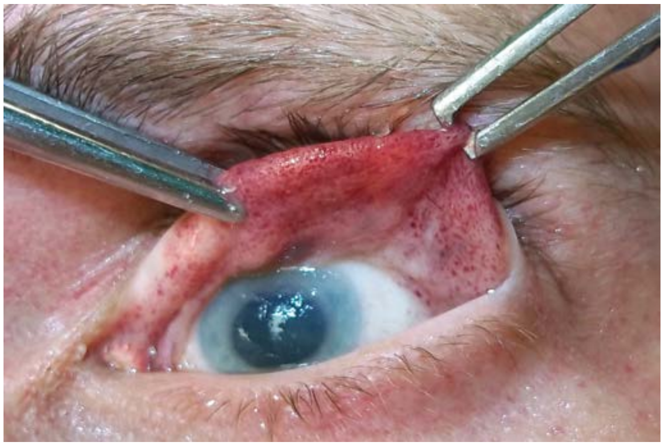

**Figure 21.10** Petechiae in the conjunctiva of an upper eyelid (incomplete hanging).

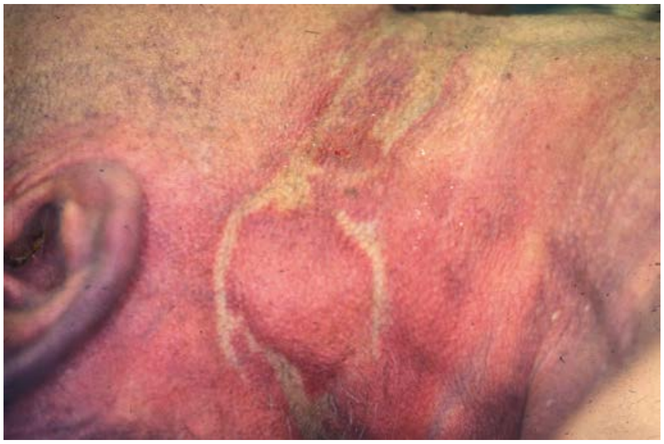

**Figure 21.12** Ligature mark with the imprint of a belt buckle (incomplete hanging using a belt).

**Ligature mark** This develops as a result of abrasion or compression of the skin by the strangulation device, which usually has a rough surface. In many cases, some sections of the mark bear an imprint of the structure of the ligature (Figure 21.12). Shortly after death, the mark is mostly visible as a pale furrow on the skin's surface. With time, the furrow dries out, taking on a brownish hue like other skin abrasions. As abrasions and desiccations may develop postmortem, the ligature mark in itself does not constitute a criterion for vital hanging [35,37]. In cases where the surface of the ligature is very smooth (e.g. a plastic-coated cable), the abrasive effect will be minimal and the mark may remain visible as a pale strip of skin or furrow for some time after death. If a large amount of material is used or if soft padding is placed under the noose, the ligature mark may merely be an impression of the folds of the cloth, or there may be no mark at all. The various ligature marks in deaths by hanging rarely present associated soft tissue haematomas [27].

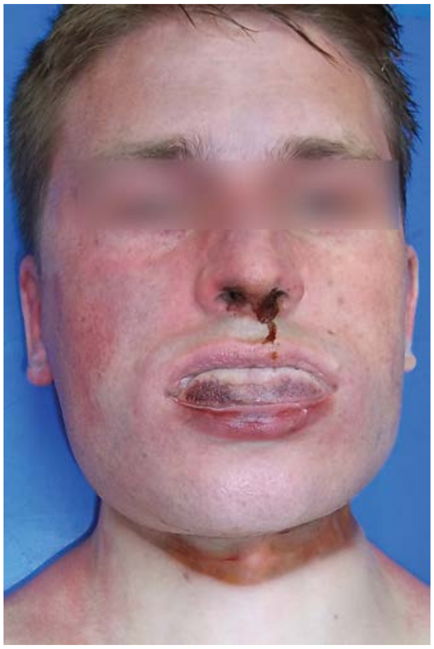

**Figure 21.11** Incomplete hanging – suicide. Congestion syndrome above the ligature mark.

In suicide hangings, the marks usually run above or around the thyroid cartilage, in cases of suicidal complete hanging, the figure is even more than 70 per cent [27].

Where the ligature encircles the neck, it will be possible to establish or reconstruct the highest point in nearly every case. In closed nooses, the highest point, also known as the point of suspension, is mostly at the site of the knot. In order to determine the highest point, it may be advantageous to measure the distance between the mark and the lower edge of the auricles. There may be no highest point in hanging deaths in a horizontal position. Very often, the ligature furrow is most prominent opposite the point of suspension.

If the noose slips upwards during the hanging, this may cause several parallel markings and broad abrasion zones that slope upwards.

A band of red skin, observed occasionally above the ligature furrow only, is an incontrovertible postmortem finding. This is a result of the blood draining from the head and pooling around the ligature. This phenomenon occurs notably in bodies that have remained undiscovered for some time [35]. In approximately a third of all cases, especially where the ligature is looped once around the neck, a continuous 'dual hyperaemia zone' may be observed directly above and below the ligature mark, which may possibly not develop postmortem [31]. Peri-ligature injuries such as skin blisters, ecchymoses or abrasions are not findings that could justify the vital origin of the ligature mark [36].

**Bleeding in skin ridges** Where a ligature is wound twice or more around the neck, overlapping folds of skin may be caught between the individual loops, known as skin

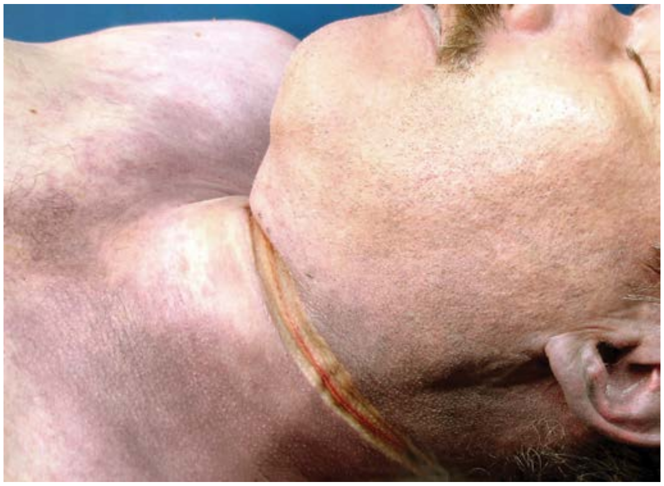

**Figure 21.13** Extravasation in the skin ridge between two ligature marks (incomplete hanging with an electric cable).

ridges. Fine red lines frequently develop in these skin ridges and are caused by extravasated blood. These are referred to as bleeding in the skin ridges (Figure 21.13). It was established as early as the 19th century that this is not a vital phenomenon [37], since the blood may also be forced out of the upper section of the skin folds in a continuous line as a result of postmortem tightening of the ligature. Hypostasis may intensify development of this phenomenon.

**Skin blisters** Occasionally, blisters filled with serous fluid may form in the interior and the periphery of ligature marks. Again, it has long been established that this is a postmortem phenomenon [37,38].

**Tracks of dried saliva** From time to time, the excessive salivation caused by hanging [39] produces dried saliva tracks in the mouth region. In some cases, the saliva drips down onto the clothing and dries. Saliva tracks may also be observed in other causes of death where the torso of the decedent was in an upright position. This finding may also occur where bodies are placed in a hanging position. Therefore, this is neither a vital sign nor is it specific to deaths by hanging [35,40].

**Protrusion of the tongue** Where the ligature passes above the larynx, the hyoid bone is pushed obliquely backwards, together with the base of the tongue, resting against the posterior wall of the pharynx. This leads to an obstruction of the airways. The tongue is then frequently pushed forward in the open mouth, protruding between the two rows of teeth. It has long been known that this finding does not represent a vital sign [35].

**Indication of a self-rescue attempt** In rare cases, victims are found with fingers trapped between the noose and the neck. This is sometimes construed as a self-rescue attempt. However, given the loss of consciousness that hanging causes after just a few seconds, the option of self-rescue is highly unlikely, thus alternative interpretations should also be considered. Therefore, this finding should not be accorded any relevant significance in evaluating the circumstances.

**Abrasions in various regions of the body** These are caused by the victim knocking into objects during the hanging process. The injuries may be expected to occur specifically during the convulsion stage. For the most part, the abrasions do not exhibit a surrounding haematoma. They cannot be distinguished from abrasions that may have been caused by cutting down or transporting the body.

**Decapitation** Thin, strong ligature materials such as plastic cables or wires contribute to this rare occurrence. In cases of incomplete decapitation, the skin of the neck may not tear apart while the internal structures are completely lacerated. In complete decapitation, the edge of the wound is predominantly clean cut similar to sharp force trauma. There may be an abrasive collar around the margins [33].

### Internal findings

**Fractures of larynx and hyoid bone** Numerous studies have been carried out on the frequency of these fractures, with highly diverse results and inadequate comparability [41]. First, the discrepancies are due to the predominantly retrospective, and rarely prospective, nature of the examinations. Second, the dissection techniques were not consistent. Occasionally, fine dissection was performed after the autopsy. In some cases, distinctions were made between complete and incomplete hanging, yet not in others.

It is an undisputed fact that thorough dissection will identify fractures to the superior cornua of the thyroid cartilage in up to 55 per cent of cases. By contrast, the cornua majora of the hyoid bone are only fractured in a maximum of 42 per cent. The thyroid cartilage laminae and cricoid cartilage are each affected in less than 10 per cent of cases [42]. Usually only one superior cornu of the thyroid cartilage is fractured (Figure 21.14). Fractures of both superior cornua of the thyroid cartilage represent the commonest combined injury. The fractures are rarer in adolescents than in the elderly, where the originally cartilaginous structures are frequently ossified [27,31].

Haemorrhages around the laryngeal and hyoid fractures are frequently scarce or even non-existent. This is evidently due to the severe compression of the blood vessels brought about by strangulation. Only in cases where pronounced haemorrhages can be ascertained may the fractures be taken to have developed antemortem. Postmortem experiments produced isolated fractures with concurrent extravasations, which were evidently indistinguishable from vital haemorrhages [40]. The evaluation must take into account the fact that fractures may also be caused

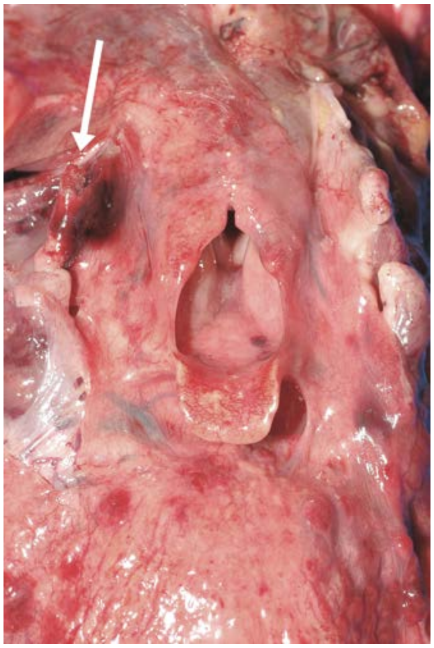

**Figure 21.14** Fracture of the right superior cornua of the thyroid cartilage with haemorrhage (incomplete hanging).

by emergency intubation. It is usually not possible to differentiate between pre-existing lesions and injuries caused by resuscitational measures [43].

**Petechiae** Particularly in incomplete hanging, petechiae may be established under the fasciae of the musculi temporales, occasionally also in the hyoid area. Rasmussen et al. [44] discovered petechiae of the tympanic membranes on a patient after attempted suicide by hanging.

**Haemorrhages in the origins of the sternocleidomastoid muscles** The haemorrhages are mostly to be found on the clavicles, more rarely on the manubrium sterni. They may be present on one or both sides and can be up to several centimetres in length. For the most part, they are more acutely pronounced on the side on which the ligature's highest point is located (Figure 21.15). Dissection of the clavicular periosteum will detect haemorrhaging in up to 96 per cent of cases of complete and incomplete hanging [45]. If the tendons at the origin of the sternocleidomastoid muscles are simply detached and no dissection of the periosteum is performed, the haemorrhage detection rate is significantly reduced [27,31,46].

The haemorrhages are a typical finding in deaths by hanging; only rarely are they observed in low intensity in other causes of death [45]. They are attributed to the impact of the forces applied to the neck during hanging, e.g. a wrenching mechanism. However, as early as 1935, Walcher [50] recognized that it is possible to produce similar extravasations postmortem. This finding was subsequently corroborated in experiments, which concluded that extravasations could still be produced on the clavicles and sternum 2–6 days after death. In these experiments, the subjects, whose cause of death was not hanging, were placed in complete hanging positions, some for a mere 10 minutes [40]. Thus, origin haemorrhages are of limited value in determining death by hanging.

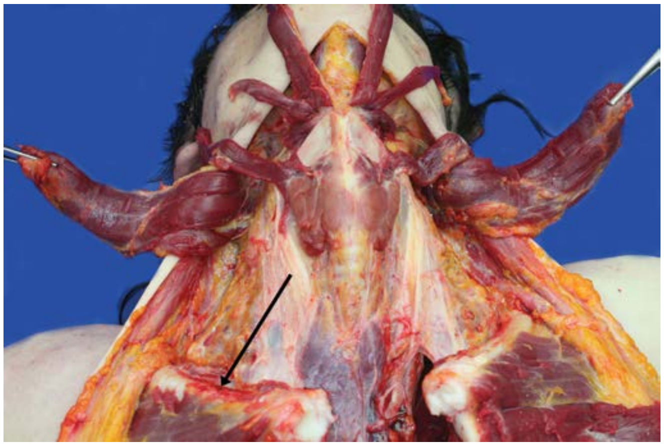

**Figure 21.15** The arrow indicates haemorrhage on the clavicular origin of the right sternocleidomastoid muscle. Layer-by-layer dissection of the anterior soft tissues of the neck (incomplete hanging).

**Intimal tears of the common carotid artery** In less than 20 per cent of cases, uni- or bilateral transverse intimal tears can be observed, which are predominantly located slightly below the carotid bifurcation [27,47]. The tears may be minimally extravasated. For the most part, the finding is attributed to the extreme tensile loading in combination with direct compression on the neck [48,49]. The tears can also be observed when jumping or falling from height into the noose (see 'External findings', Decapitation).

On very rare occasions, the tears also occur in a longitudinal direction. The finding does not represent a vital sign [50].

**Other injuries to the soft tissues of the neck** Haemorrhages in the subcutaneous tissue, the other neck muscles or the laryngeal mucous membrane are infrequent. In up to 34 per cent of cases, extravasations were found in the muscles of the larynx. The ligature furrow itself exhibits minimal perfusion [27,35,50]. Isolated haemorrhages can be ascertained behind the pharynx and in the prevertebral tissue. In addition, haemorrhages may occasionally occur in the nuchal muscles, evidently caused by the wrench to the neck. Thorough dissection of the larynx will enable haemorrhages in the capsules or the spaces of the cricoarytenoid articulation and the cricothyroid articulation to be demonstrated. In the course of systematic studies, these haemorrhages were recorded in less than half of all cases [31].

In extreme incidences of trauma, as in falls from a height with the noose around the neck, ruptures of the fasciae and the neck muscles may be observed. As a general rule, associated haemorrhages are only minimal or non-existent [33].

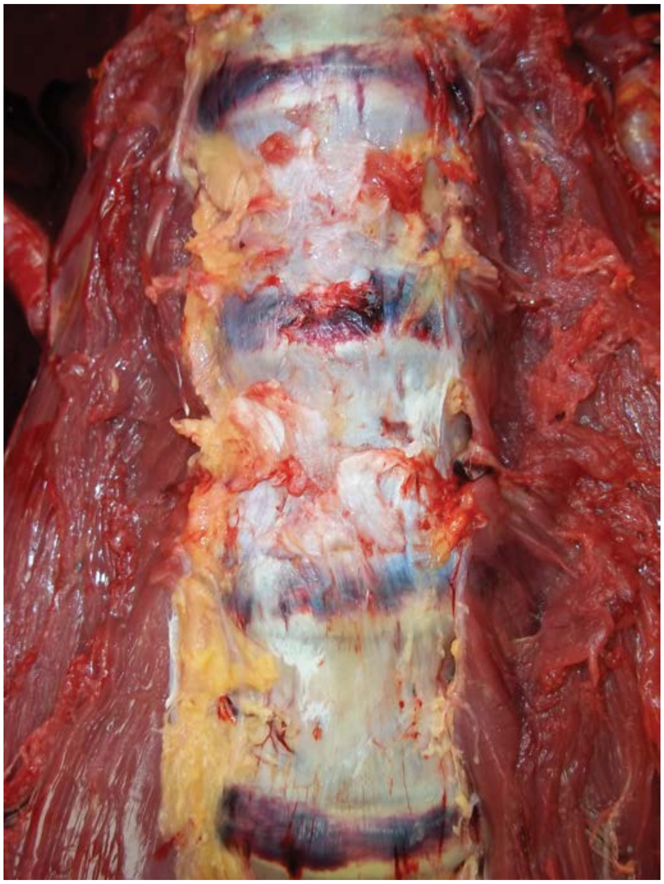

**Figure 21.16** Haemorrhages on the front of the intervertebral discs of the lumbar spine (incomplete hanging).

**Haemorrhages on the front of the intervertebral discs** These are fine streaks of haemorrhages, predominantly found on the ventral surfaces of the intervertebral discs of the lumbar spine (Figure 21.16). First described by Simon in 1968 [51], these are also called Simon's bleedings. They are occasionally present in the upper sections of the spinal column. In adolescents and young adults, the finding may be of extremely high intensity, particularly in complete hanging. The haemorrhages can be found in over 40 per cent of all deaths by hanging [31,52]. The causative mechanism would appear to be a combination of the traction and spasms produced by hanging. In particular, these intervertebral haemorrhages also occur as a result of intensive blunt force trauma with hyperextension and/or hyperflexion of the spinal column (e.g. in road traffic accidents). Where putrefaction is established, the finding may be simulated due to haemolysis [52,53]. Postmortem suspension did not produce comparable extravasations [40]. Nowadays, Simon's bleedings are deemed to be a vital sign in death by hanging if the other eventualities mentioned above can be ruled out.

**Fractures of the cervical spine** This type of injury is only observed in exceptional cases of suicide hangings. As a general rule, fractures of the first to third cervical vertebrae only occur after a fall or jump from a certain height with the noose around the neck [21,54]. Details on the special form of hangman's fracture can be found elsewhere (see Section 2, 'Hangman's fracture'). The anatomical conditions are such that fractures of the second cervical vertebra are not easy to diagnose during autopsy. A computed tomography (CT) scan carried out prior to the autopsy facilitates the diagnostic investigation.

Fractures of the lower cervical and upper thoracic spine represent an anomaly in deaths by hanging and are sometimes a consequence of pre-existing conditions [55].

**Bowel wall haemorrhage** Haemorrhages in various sections of the intestinal wall have been reported in cases of hangings [56,57]. The finding was found in just over 10 per cent of cases. The extent to which the finding is strongly associated with the diagnosis of hanging requires further investigation.

**General findings** Uncoagulated blood in the heart and hyperaemia of the internal organs are regularly available. Acute pulmonary emphysema and cerebral oedema can be diagnosed frequently.

## Differentiation between suicide, homicide and accident

### Suicide

It must be evaluated whether there are any grounds to indicate foul play. The ligature submitted must be adequate to account for the ligature mark and any other abrasions on the neck. The assessment of vitality in hanging may only rest upon several findings characteristic of hanging, and not merely on a single finding. The pattern of findings should be considered in the context of the overall circumstances. Furthermore, the inner sides of both arms should always be dissected to eliminate the presence of any haematomas due to finger marks. As a general rule, where the remains are in an advanced stage of putrefaction, it is not possible to definitively rule out any external intervention. The determination of blood-ethanol concentration and toxicological examinations are essential for the evaluation [58]. If there are no inconsistent findings, the hypothesis of suicide may be assumed, based on the forensic evidence. In rare cases, the conclusion that it is indeed suicide is supported by the evidence of recent self-inflicted injuries such as practice slashes on the wrists. Byard [59] reported a case of suicidal hanging that revealed obvious self-injuries to the scrotum and rectum. In isolated cases, a suicide hanging may be unsuccessful, for example due to the ligature breaking. The suicide may remain capable of action and even leave the scene, with death not occurring until sometime later, in some cases as a result of a second suicide situation [60,81]. The circumstances of the death subsequently give rise to suspicions of external intervention. Evaluation of such cases is only possible on the basis of the medical and toxicological findings, the results of the reconstructions and the police investigation.

## Homicide

Where the hanging was the sole intent of the foul play, the body of the unsuspecting victim bears very little evidence of a struggle. Consequently, the constellation of the findings could also be attributed to suicide. Such cases are, however, almost unheard of. As a rule, they involve defenceless victims such as small children, the infirm or the elderly. In addition, victims of such homicides may also include persons who, owing to intoxication, are largely or wholly incapacitated. Defenceless victims may be pulled up using the ligature or alternatively pushed from a height with the noose around their necks [23].

Occasionally, foul play may lead to death by hanging where previous trauma did not prove fatal. For the most part, the additional injuries that are not characteristic of hanging will cause external intervention to be suspected [61]. Nevertheless, doubts may arise, particularly if the primary trauma was directed towards the neck. It may prove difficult or even impossible to differentiate between previous trauma and subsequent findings from death by hanging. The presence of relevant haemorrhages in the soft tissues of the neck, which are hardly ever found in cases of hanging, is invariably an indication of additional trauma to the neck. Fine dissection may yield additional evidence of this. For example, injuries to the cricoid cartilage and the upper trachea are a constellation of findings that is rarely observed in deaths by hanging.

Finally, a few extremely rare cases have been reported of the hanging of victims who had previously been killed by a different method, in order to simulate a suicide. The actual cause of death may be ligature strangulation or intoxication, for instance. Admittedly, the postmortem injuries to the neck may closely resemble the few vital findings that are characteristic of hanging [40]. An assessment of vitality may well prove impossible in such cases. Where traces of further trauma are discernible (e.g. an additional ligature furrow or manual strangulation marks on the neck), these may be evidence of a previous homicide. Attention should be paid to injuries that could not have been caused by the body knocking into objects during the hanging process (e.g. finger marks and transportation injuries). Furthermore, the circumstances at the scene of death must be taken into consideration. A congestion syndrome would be inconsistent in cases of complete hanging, therefore a previous throttling incident must be taken into consideration. If no petechiae have developed in cases of incomplete hanging, this also represents an abnormality. Moreover, it should be noted that incomplete hanging and ligature strangulation may result in extremely similar patterns of finding. If no differentiation is possible, the police and public prosecutor must be notified. The final evaluation of such cases may only be carried out once all the facts are known, paying particular attention to the results of the chemical and toxicological tests.

## Accident

Accidental hangings are extremely rare. Most accidents occur in near-hangings intended to enhance sexual stimulation, which can be considered a paraphilia [2,19]. For the most part, accidental deaths can be deduced from the circumstances, including any clothing and accessories used, along with other characteristics that serve to indicate a sexual context. Occasionally, subjects may record videos or take photographs of their sexual practices. If there is no conclusive indication of a sexual context, it may be difficult or even impossible to define the boundaries of the suicide. It should be noted that, in cases of autoerotic activity, mechanisms that facilitate near-hanging are generally deployed (e.g. in the form of coil springs or elastic bands as part of the ligature).

Other accidents caused by hanging that may occur at random in everyday situations are extremely rare [62]. Most of these involve a fall resulting in an incomplete hanging

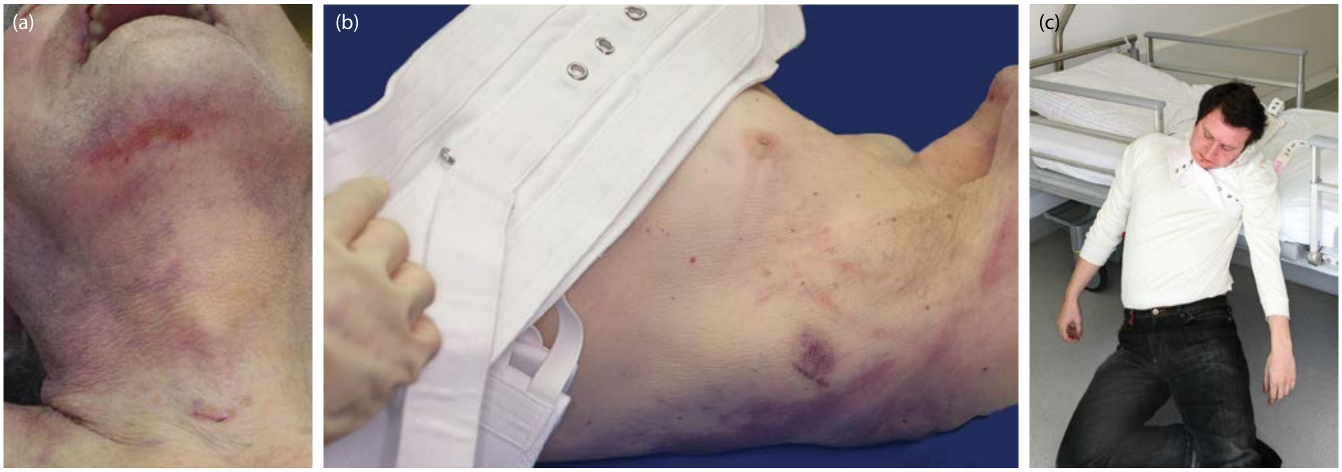

**Figure 21.17** A patient had fallen out of bed while restrained using an abdominal strap. The sequence of events was initially unclear. (a) Abrasion under the lower jaw, small wound under the larynx, both on the right. (b) Patchy and linear haematomas under the left armpit. (c) Reconstruction of events and the scene of death by a stuntman. Cause of death: incomplete hanging.

situation; even accidental complete hanging has been known. Corresponding fall-related injuries may be present on other parts of the body apart from the neck. Strangulation is effected by means of ligature materials that function like an open noose. On occasion, the strangulation devices may be hard objects such as boards, beams, the edges of wash basins and forked branches [63]. These objects may cause extensive ligature abrasions. Such accidents involve persons who are unable to free themselves, especially infants and small children. In isolated cases, fatal hangings occur in children's playgrounds where nets are stretched over the climbing frames. In the event of a fall, the head or a bicycle helmet strap may get caught in the net, leading to death by hanging [26]. In rare cases, comparable accidents involving adults have even been recorded [21].

Accidental hangings also occur in nursing, including geriatric nursing, where strap restraints are not affixed correctly and the patient falls out of bed. The victim is later discovered hanging from the bed with the strap around their neck. In order to avoid danger of strangulation, special regulations have been set up regarding the use of strap restraints. The autopsy may detect very few signs of injury in such cases (Figure 21.17), therefore the cause of death may be determined only after a reconstruction of the event [20,64].

## 21.4 Ligature strangulation

■ *Wolfgang Keil*

### Definition

Ligature strangulation is a fatal compression of the neck by means of a strangulation device, which is usually tightened using the hands, or in rare cases, by an object or machine.

### Frequency/occurrence

Relatively few cases of death by ligature strangulation are recorded. The majority of cases are due to foul play. Evidence of ligature strangulation is frequently detected in conjunction with other traumatic violence such as manual strangulation and blows [65]. Ligature strangulation may therefore be either the eventual or a concurrent cause of death. Evidence of ligature strangulation on the decedent may constitute only a secondary finding. The number of non-fatal ligature strangulations due to foul play is estimated to be comparatively high. Suicidal or accidental ligature strangulation is rare.

### Strangulation devices

The basic characteristics of the strangulation devices deployed in ligature strangulation are essentially the same as those used in hanging. In rare cases, the victim's necklace is used as a ligature. One case of ligature strangulation using hair extensions has been reported [66]. Occasionally, items of clothing worn around the victim's neck are used as ligatures. In suicide by ligature strangulation, rubber bands and other elastic materials are also used. Kumar et al. [67] published a suicide in which a plastic lock tie was used for neck compression. Also, the contraction of a noose around the neck by a tethered stone was observed in the case of a suicide [68]. Strangling mechanisms may be wound once or more round the neck. Ligature strangulation by means of hard objects such as metal loops or iron bars is termed 'garrotting'. The word comes from the French word *garrotte*, a metal device used to compress the neck.

### Classification of the circumstances

In fatal attacks entailing ligature strangulation alone, it is usual to find no signs of a struggle in the vicinity of the victim. It should be noted that cases of ligature strangulation involve victims who are asleep, defenceless or significantly inferior in terms of physical strength. By contrast, signs of a struggle are generally to be expected where ligature strangulation was the final act in a multi-phase incident. In isolated cases, the perpetrators attempt to simulate a suicide after strangulation. To do so, the victim is suspended, for example, or a plastic bag is placed over their head and tied around the neck over the ligature mark. The circumstances at the scene of a suicidal ligature strangulation may not differ from that involving a third party. Although extremely rare, accidents mainly occur when an item of clothing worn on the neck or shoulders of the victim gets caught in engine-driven machine parts (e.g. in escalators or conveyor belts).

### Pathomorphology

#### External findings

**Petechiae/congestion syndrome** A congestion syndrome usually develops in both fatal and non-fatal ligature strangulation. It is generally more intense than in other forms of strangulation [69] because the strength applied by the hands and arms in tightening the ligature merely leads to a compression of the veins, at least in adult victims. Even if the arteriae carotides are compressed, the arteriae vertebrales appear to remain open in many cases. The congestion develops above the ligature mark (Figure 21.18). The petechiae occasionally merge in the skin, creating small haemorrhagic areas. It is not uncommon to find hyposphagma (Figure 21.19) or, in some cases, bleeding from the nose, more rarely from the ears [70]. A comparable intensity of congestion is only observed in exceptional cases of incomplete hanging. The face becomes cyanotic due to the rapid deoxygenation of the haemoglobin in the process of the strangulation. Frequently, a swelling of the face may be observed when serum is pressed into the soft tissues as

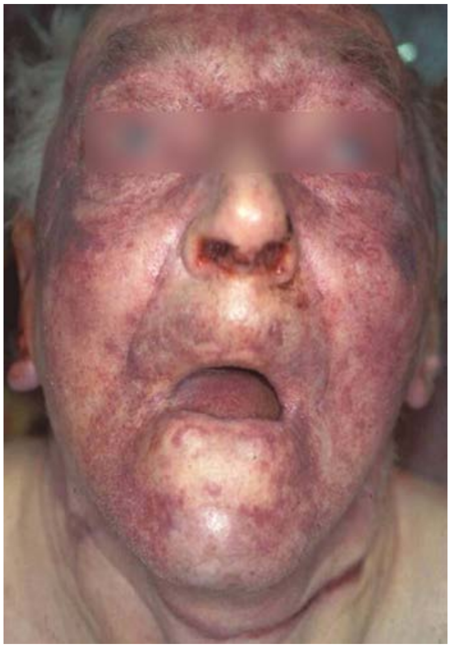

Figure 21.18 Ligature strangulation – homicide. Intensive congestion syndrome above the ligature mark, bleeding from the nose due to congestion.

a result of the stasis. A few notable exceptions are when great force is applied to strangle newborns, infants or small children. Since this enables the simultaneous compression of all neck arteries, only minimal congestion syndrome may develop, if any [69,70]. If, during an attack, initial sharp or blunt force trauma results in open wounds in the head and neck area, any subsequent ligature strangulation will not produce intensive congestion while the wounds are bleeding; thus, very few petechial haemorrhages will develop [71].

**Ligature mark** Like the mark produced in death by hanging, this is caused by the abrasion of the ligature on the skin. If the ligature is a wide band of cloth with a smooth surface, the lesion of the stratum corneum may be so minimal that no mark is discernible. Non-fatal ligature strangulations may simply leave a reddish hyperaemia on the skin of the neck; there may be no ligature mark at all. In the majority of cases, the ligature furrow runs horizontally around the neck, meaning that no highest point can be established or reconstructed [70].

Exceptions to this rule are when a person is strangled while sitting or lying down and the perpetrator is standing over the victim. Slanting ligature marks exhibiting a highest point may be observed in cases where there is a large difference in height between the victim and the perpetrator (e.g. between adults and children). Where the ligature mark shows an upwards deviation, it is important to bear in mind that the tension on the neck may result in a partial hanging mechanism. In turn, this may cause abrasions on the neck due to the ligature slipping upwards. The ligature mark is not a vital sign. However, on occasion there may be ambient haematomas, which are indications of the vital character [70].

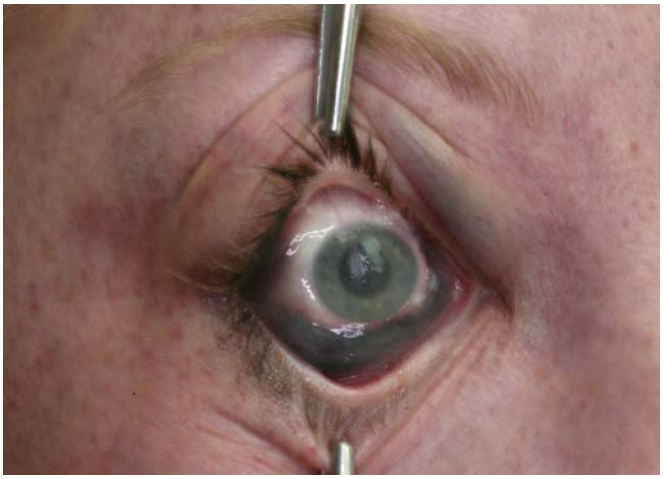

Figure 21.19 Ligature strangulation – homicide. Hyposphagma.

## Internal findings

**Fractures of larynx and hyoid bone** As with the other strangulation mechanisms, the proportion of fractures detected depends on the dissection technique. Haarhoff [70] was unable to verify fractures to the larynx or to the hyoid bone in 40 autopsies of fatal ligature strangulation. Koops et al. [72] identified fractures to the larynx or the hyoid bone in 66 per cent of cases of homicidal ligature strangulation. In his autopsies, Maxeiner [73] established fractures in around 43 per cent of cases following fine dissection of the larynx. Bilateral fractures to the superior cornua of thyroid cartilage were by far the most frequent finding. Cricoid cartilage fractures are extremely uncommon. In rare cases, the hyoid bone is also fractured. Due to the intensive congestion syndrome, the fractures are generally a great deal more perfused than in death by hanging. By contrast, haemorrhaging in the joints of the larynx was determined in less than 10 per cent of cases [74].

**Petechiae/congestion syndrome** Any petechiae generally occur in the scalp, under the fasciae of the musculi temporales (Figure 21.20) and under the pleura pulmonalis.

**Haemorrhages in the neck structures** In approximately half of all cases, soft tissue haemorrhages are established that correspond to the furrow of the ligature. The haemorrhages are located in the subcutaneous tissue, under the muscle fasciae and in the musculature itself [70,75]. They are usually less intensive in the nape than at the front and lateral areas of the neck. There is substantial bleeding in the soft tissues of the larynx and particularly in the posterior cricoarytenoid muscle.

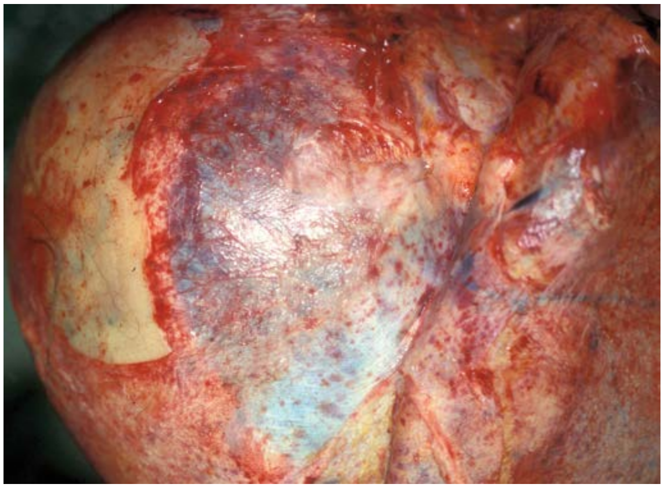

**Figure 21.20** Ligature strangulation – homicide. Petechiae under the fascia of the right musculus temporalis.

**Haemorrhages in the origins of the sternocleidomastoid muscles** This finding is infrequently observed, notably in cases where tension has been applied to the neck during strangulation. Therefore, where the ligature mark slants upwards, it is imperative that any origin haemorrhages be taken into consideration.

**Tongue haemorrhages** With its multitude of separate veins, the tongue is located in the anatomical vicinity of the venae jugulares internae. Thus, congestion in the head and neck areas inevitably causes haemorrhaging of the tongue. First, submucosal haemorrhages, a common occurrence as part of congestion syndrome, are almost invariably located at the base of the tongue. Second, very intensive haemorrhaging often develops intramuscularly. The horizontal cross section of the tongue discloses linear streaks, occurring mainly in the middle and posterior third (Figure 21.21). These intramuscular haemorrhages are known as 'apoplexy of the tongue'.

**Formation of foam in the airways** Occasionally, a fine, white foam may adhere to the laryngeal, tracheal and bronchial walls, which may be streaked with blood [75]. The foam evidently accumulates from bronchial secretion and tidal air during dyspnoea. This is possible because, although the larynx is compressed during ligature strangulation, it is not completely closed in most cases. In this regard, perpetrators occasionally report inspiratory stridor of the victim during ligature strangulation.

**Acute pulmonary emphysema** Observed from time to time, this finding is a result of the increase in respiratory resistance during the compression of the airways.

**General findings** Uncoagulated blood in the heart, acute hyperaemia of the internal organs and acute pulmonary emphysema. Cerebral oedema can be considerable.

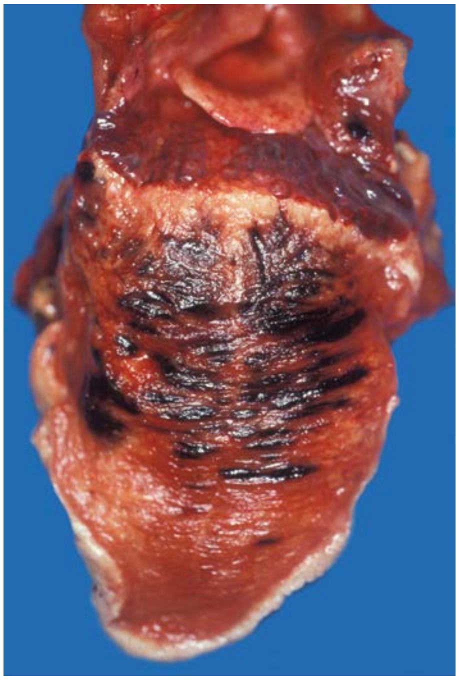

**Figure 21.21** Ligature strangulation – homicide. Intensive haemorrhages in the middle and posterior third of the tongue.

## Differentiation between suicide, homicide and accident

### Suicide

The strangulation device is still in place. As a general rule, the suicide winds it around the neck several times. On occasion, the ligature is knotted at the front of the neck. Sometimes suicides insert objects under the loops in order to increase the compression pressure. In rare cases, one end of the rope is tied to a higher point and the other wound around the neck so that suicide by ligature strangulation may end in death by hanging. If clear congestion syndrome is present, there can generally be no doubt as regards the vitality of the events. The hypothesis that states that laryngeal and hyoid fractures are less common in suicidal than homicidal ligature strangulation is evidently unsubstantiated; therefore, it is irrelevant in evaluating individual cases [73]. As regards the possibility of external intervention, the principles laid down for the evaluation of suicide hangings also apply here (see 21.3, 'Hanging').

### Homicide

In many cases, the perpetrators remove the ligature from the victim's neck after death. A single loop of the strangulation ligature is very common in homicides. Where the ligature is knotted at the back of the neck, foul play must be suspected. In many cases, external intervention is immediately evident because fatal and non-fatal ligature strangulation frequently involves other traumatic violence [70,73]. This normally results in injuries that are inconsistent with the hypothesis of suicide and thus are clear evidence of foul play. Remarkably, one case of iatrogenic ligature strangulation due to a misplaced bandage in the head and neck region has been reported [76].

### Accident

As a general rule, the circumstances are a clear indication of the accidental nature of such infrequent events. In exceptional cases, accidents may also happen as a result of unusual sexual practices. For example, where bondage involves tying a person's bent legs and neck together using physical restraints while in a lying position, the tension on the neck can lead to accidental self-strangulation.

# 21.5 Manual strangulation

■ *Wolfgang Keil*

### Definition

Manual strangulation is a fatal compression of the neck caused by applying direct pressure of the hands.

### Frequency/occurrence

Fatal cases of manual strangulation are rarely encountered in forensic pathology practice. Several studies indicate that deaths by manual strangulation may be slightly more common than by ligature strangulation [70,73]. According to investigations by DiMaio [24], victims are more likely to be female than male. Manual strangulation invariably represents external intervention and is often observed in conjunction with other traumatic violence [65]. Where a multi-phase incident entails varying types of violence, manual strangulation may be either the actual or a concurrent cause of death. In some cases, evidence of manual strangulation may constitute a secondary finding, with the attack, which the victim survived for a certain period of time, occurring prior to the actual homicide.

Non-fatal cases of manual strangulation are observed relatively frequently, some in association with sexual offences. It is important to bear in mind that death by manual strangulation may occur as a result of sadomasochistic sexual practices [65].

### Classification of the circumstances

In the event of defensive action where the perpetrator and victim are evenly matched in terms of physical strength, the strangulation process may last for some time until death eventually occurs. Accordingly, the homicide scene may provide indications of a struggle. The perpetrator often uses one hand to combat resistance or immobilize the victim, while squeezing the throat with the other hand. In some cases, the perpetrator ultimately kneels or sits on his victim to hold him down in order to complete the strangulation process. Occasionally, the victim is found in this final position.

## Pathomorphology

### External findings

**Petechiae/congestion syndrome** Acute congestion develops to a greater or lesser extent, depending on the variation of neck holds, in the head and neck area. Congestion syndrome occurs in most cases as the compression of the venae jugulares with simultaneous arterial supply interrupts venous drainage, if only temporarily. As a general rule, congestion syndrome is less intensive in fatal manual strangulation than in ligature strangulation [69] and haemorrhages from the nose and ears are hardly ever observed. However, when comparing cases of death by manual and ligature strangulation, Haarhoff [70] found no relevant differences in the location and intensity of petechial haemorrhages. Especially when the larynx and the trachea are compressed during manual strangulation, the resulting hypoventilation may be the determining factor for the onset of death. Thus, the congestion is then clearly less pronounced. In rare isolated cases, in which the carotid sinus reflex may be a contributing factor, manual strangulation does not cause petechial haemorrhages [77]. When large hands apply pressure to a slender neck, petechiae may also be minimal or non-existent simply because the arteriae carotides are compressed.

**Manual strangulation marks** The formation of marks depends to a large extent on the type of attack. In most cases, manual strangulation takes place from the front, in rare cases from the side or behind. Strangulation may be carried out by applying only one or both hands. Accordingly, strangulation marks are exhibited not merely in the laryngeal area but also below the lower jaw, on the lateral areas of the neck, rarely in the nape of the neck. It is important to differentiate between two types of strangulation marks that frequently occur in combination: first, there may be fingernail impression marks, which are visible as short, curvilinear or S-shaped abrasions of the skin (Figure 21.22). If the perpetrator has short fingernails or wears gloves, these findings may be absent. Second, haematomas caused in particular by the pressure of the finger pads may frequently be evident on the skin of the neck. These haematomas are likewise an indication of the vitality of the neck compression. According to the results of studies in 40 homicides, the positioning of the strangulation

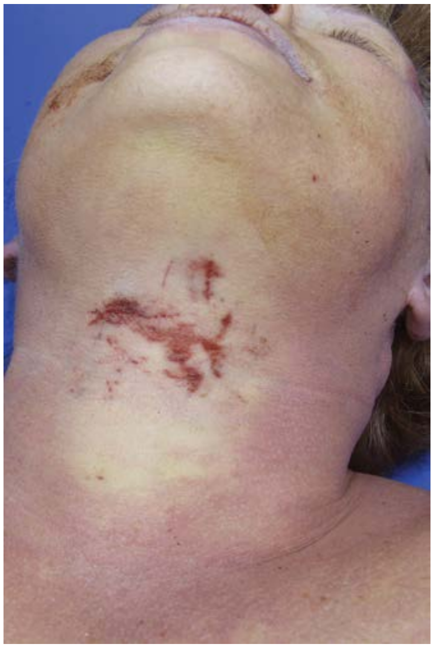

**Figure 21.22** Manual strangulation. Fingernail impression marks.

marks do not permit any conclusions to be drawn regarding the course of events [70]. In living victims, strangulation marks may merely consist of erythema, which fades after a few hours.

### Internal findings

**Fractures of the larynx and hyoid bone** In up to 80 per cent of cases, fractures may occur that are clearly perfused to a large extent. The injury rate is therefore considerably higher than in ligature strangulation. The superior cornua of the thyroid cartilage are most commonly affected, the cornua majora of the hyoid bone less so [24,70,73]. Fractures of the thyroid cartilage plate and the cricoid cartilage are rare, but they occur more frequently in manual strangulation than in ligature strangulation. Nevertheless, it is not possible to differentiate between manual and ligature strangulation on the basis of the fracture pattern [73]. Lesions in the joints of the larynx may be observed more frequently in manual than in ligature strangulation [74].

**Petechiae/congestion syndrome** As in ligature strangulation, petechiae are a common occurrence of congestion syndrome in the scalp, in the musculi temporales and under the pleura pulmonalis.

**Haemorrhages in the soft tissues of the neck** These are particularly likely to develop in the proximity of the larynx but may be found in all areas and layers of the neck structures. Moreover, haemorrhages occasionally develop under the capsules of the thyroid lobes. Studies have shown that haemorrhagic intensity and distribution are frequently more pronounced in manual strangulation than in ligature strangulation. In many cases, haemorrhages are disclosed during dissection of the nuchal muscles.

**Haemorrhages in the origins of the sternocleidomastoid muscles** This finding is rare in manual strangulation, apparently because, in most cases, less pulling force is applied to the neck than in other forms of strangulation.

**Formation of foam in the airways** As in ligature strangulation, a fine, white foam, sometimes streaked with blood, may occasionally adhere to the laryngeal, tracheal and bronchial walls.

**Acute pulmonary emphysema** This type of emphysema may occur in varying degrees of intensity.

**General findings** Comparable with the findings in ligature strangulation, i.e. uncoagulated blood and acute congestion of internal organs. Furthermore, there is a more or less pronounced pulmonary emphysema and often a significant cerebral oedema.

## Differentiation between suicide, homicide and accident

### Suicide

Suicide by manual strangulation is impossible since the onset of unconsciousness decreases muscle tone and the hands stop applying pressure to the neck. In rare circumstances, as part of various forms of self-inflicted injuries, manual strangulation marks may be observed on the skin of the neck.

### Homicide

Where fingernail impressions and/or haematomas in the form of strangulation marks are established on the neck, foul play must always be assumed as the primary cause. The presence of petechiae signifies that external intervention is beyond doubt. If there are no strangulation marks yet the head and neck area exhibits petechiae, manual strangulation must be considered in addition to ligature strangulation. Since the victim is likely to resist manual strangulation, signs of a struggle may well be manifest on the body, which also point to foul play. Attention should be paid to potential counter-pressure injuries on posterior parts of the body if the victim has possibly been pinned down on the ground. These injuries develop particularly in prominent areas of the back and buttocks. Since manual strangulation is also practised to enhance sexual stimulation, this eventuality must also be considered in isolated fatal cases.

### Accident

Accidental fatal and non-fatal manual strangulation must always be eliminated.

## 21.6 Other compressions of the neck

■ *Wolfgang Keil*

### ■ Neck holds

#### Definition

Neck compressions using an arm are known as forearm choke holds, whereby the hand of the free arm may be applied to increase the compression of the neck.

#### Forms

There are two distinct modi operandi, namely choke holds and carotid sleeper holds [78]. In practice, a combination of both techniques is common. On the one hand, this is because victims put up resistance, twisting and turning to escape from the neck hold. On the other hand, the assailants do not apply the holds in a professional capacity.

#### Choke holds

One arm is placed around the neck from behind the victim. The bent forearm runs across the front of the victim's neck, applying pressure and constricting the larynx and/or the upper trachea. The free hand can increase this pressure by pulling the wrist of the forearm backwards, thereby sealing the airways. At the same time, venous congestion may develop in the head area. As a general rule, unconsciousness sets in after several minutes.

#### Carotid sleeper holds

In this restraint technique, the front of the victim's neck is in the crook of the assailant's elbow, thus there is very little pressure on the larynx and trachea. In particular, the lateral areas of the neck with the arteriae carotides are constricted by the pressure of the forearm and the counter-pressure of the upper arm. The free hand may be used to significantly increase this pincer effect. When minimal pressure is applied, bradycardia and tachycardia are observed, which may be classified as an indication of the effects of pressure or decompression on the carotid sinus. If enormous pressure is applied, this may even lead to compression of the arteriae vertebrales before they enter the transverse processes of the cervical spine or in the area of the atlas loop. This effect is similar to neck compressions that are carried out with blood-pressure cuffs. Carotid sleeper holds may result in loss of consciousness in less than 15 seconds [79].

#### Frequency/occurrence

Forearm neck holds are frequently used every day in physical altercations. For the most part, choke holds are applied. They may potentially prove life-threatening if the neck is intensively compressed for several minutes or if arterial obstructions develop as a result of extreme lateral pressure. This can render subjects unconscious. However, fatalities have only been reported in extremely rare instances [78]. Furthermore, these choke holds are an integral part of certain martial arts that include an element of self-defence. These sports involve predefined neck holds, notably the carotid sleeper hold. There are no reports of fatalities as a result of neck holds in martial arts. The carotid sleeper hold is also an integral part of the restraints deployed by the police, law enforcement officers and guards. Several deaths have been linked to these restraint measures [78]. In principle, the use of neck holds by police officers would appear to be indispensable in certain circumstances. However, it is essential that officers receive proper training on how to carry out the holds correctly. Under no circumstances should techniques that could cause arterial compression be applied for longer than 10–15 seconds in order to avoid any life-threatening incidents.

#### Pathomorphology

##### External findings

**Petechiae/congestion syndrome** Petechiae may be present in the conjunctivae, while congestion syndromes evidently do not occur [78,79].

**Haemorrhages in the skin below the lower jaw** These findings were observed on the edge of the lower jaw [23].

**Injuries to the skin of the neck** No such findings were reported in the cases publicised. Streaky haemorrhages may be present, possibly caused by tension applied to items of clothing [79].

**Foamy discharge from the nostrils** One of the fatalities published by Denk and Missliwetz [79] exhibited this finding.

##### Internal findings

**Fractures of larynx and hyoid bone** In each case examined, Reay and Eisele [78] established the fracture of one superior cornu of the thyroid cartilage. Denk and Missliwetz [23] did not observe any fractures in deceased subjects, however they managed to produce laryngeal and hyoid fractures on bodies in experiments using certain choke holds.

**Petechiae** These may be present in the mucous membranes of the pharynx and larynx.

**Injuries to the soft neck tissues** Minimal to intensive haemorrhages of the neck muscles may be found.

### Classification of such fatalities

The evaluation must take any risk factors of the decedent into account. These include pre-existing conditions, particularly of the cardiovascular system, convulsions and psychological disorders with manic episodes. Furthermore, acute intoxication, alcohol withdrawal, narcotics and medication may have a contributory impact on fatalities occurring during the application of neck restraints. Persons with risk factors are more vulnerable to the effects of acute cerebral $O_2$ deficiency. In addition, the carotid sinus reflex may have an extremely adverse impact where there are pre-existing conditions. Where the autopsy and further examinations can rule out these risk factors, a direct causal link between the forearm choke hold and the demise may be considered a certainty [78].

## Kneeling/placing a foot on/pressing the forearm on the neck

### Definition

Neck compression may be caused by the impact of the knee area, the feet or the forearm, whereby at least part of the assailant's body weight operates on the victim's neck.

### Frequency/occurrence

These forms of neck compression are occasionally observed in multi-phase homicides or attempted homicides. Studies have shown that the victim is first pinned to the ground in a lying position using other forms of traumatic violence. The assailant subsequently kneels or places his foot on the victim's neck. In rare cases, kicks may also be delivered to the neck. In a very small number of cases, the assailant may lean on the victim's neck using their forearm.

### Classification of the circumstances

As a general rule, there will be signs of a struggle. The victim is usually in a lying position on the ground.

### Pathomorphology

#### External findings

**Petechiae/congestion syndrome** As the neck is generally compressed from the front or one side in this type of attack, petechial haemorrhaging is relatively mild. In the event of congestion syndrome, this is generally also of minimal intensity. As regards petechiae, it should be noted that, prior to the attack with the legs or feet, neck compression may have taken place by other means (e.g. by manual or ligature strangulation).

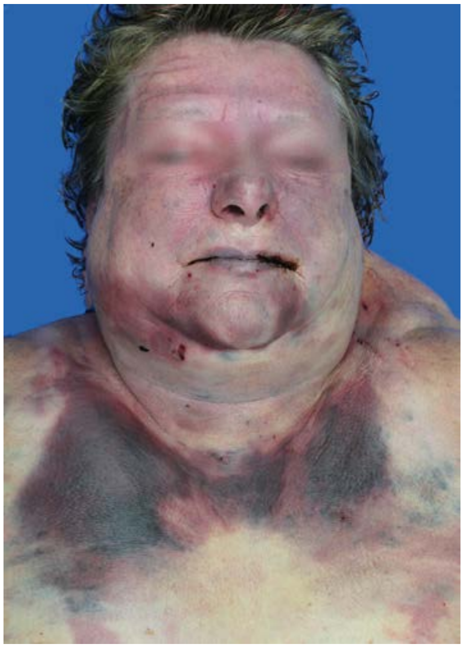

**Figure 21.23** Kneeling on the neck. Haematomas over the entire front of the neck, particularly over the clavicles, under the chin and under the lower jaw on the right. Intensive congestion syndrome.

**Injuries to the skin of the neck** Extensive abrasions and haemorrhages may be found (Figure 21.23). Nevertheless, kneeling or leaning on the victim may not invariably cause injuries that can be established externally. If pressure is applied to the neck using the feet, the resulting injuries will depend on whether shoes were worn. The type of shoe sole will be a significant factor in the formation of abrasions and haematomas. If bare feet are used to compress the neck, this may not necessarily result in any damage to the skin.

#### Internal findings

**Fractures of larynx, hyoid bone and tracheal cartilage** Due to the extensive trauma, usually of greater intensity in attacks using the knee or the feet, there may be fractures of the hyoid bone, larynx and upper tracheal cartilage, which are generally perfused to a large extent. The combination of such fractures is an indication that compression took place over a larger area. The larynx and upper tracheal cartilage may be fractured in several places, a finding rarely observed in manual or ligature strangulation. Fine dissection of these structures is indispensable in documenting the extent of the injuries. Where the assailant leans on the victim using the forearm, this does not cause such intensive fractures.

**Haemorrhages in the soft tissues of the neck** Intensive neck compression over a large area may lead to widespread subcutaneous haematomas, haemorrhages and contusions of the musculature, occasionally also of the mucous membranes. The area of injury may extend as far down as the clavicles and the manubrium sterni.

**Formation of foam in the airways** As in the other forms of neck compression, this finding may be present.

**General findings** Uncoagulated blood and acute hyperaemia of the internal organs. Acute pulmonary emphysema and brain oedema are possible.

## Differentiation between suicide, homicide and accident

### Suicide

Suicide and self-inflicted injuries are virtually impossible in this context. In theory, committing suicide by lying down in front of a car wheel is conceivable.

### Homicide

Foul play must always be assumed as the primary cause of such severe neck injuries. As a general rule, the body will exhibit evidence of a struggle. The circumstances at the scene of death will usually provide additional indications that this is a case of homicide.

### Accident

In practice, the evaluation of whether the fatality was accidental may be deduced from the circumstances. In very rare cases, it is feasible that moving machine components may have an impact on the neck, resulting in intensive compression injuries. In agricultural settings, accidents involving ungulates may also cause severe compression of the neck. However, the accidental nature is corroborated by the overall pattern of injury and the circumstances of the death.

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# 22 Traumatic, Crush and Compression Asphyxia Including ‘Burking’

*Guy N. Rutty*

## Introduction

The cells of the body require a continuous supply of oxygen to produce energy. Oxygenation of the blood requires two processes: The availability of oxygen within the breathable atmosphere at a level that can sustain life, and ventilation (breathing), i.e. the process by which oxygen is drawn from the atmosphere into the lungs where oxygen then enters the blood to be circulated around the body. Failure of oxygenation of the blood through insufficient availability of oxygen or the failure of ventilation results in hypoxia, which, in turn and depending on the circumstances, can result in morbidity and mortality (Figure 22.1).

Mechanical asphyxia is a generic term that encompasses many different mechanisms which interfere with the supply of oxygen to the lungs during ventilation. One subcategory of mechanical asphyxia is so-called ‘traumatic/crush/compression asphyxia’, which are overlapping terminologies used when the movement of the chest and/or abdomen is compromised by an external force applied to it as opposed to an internal pathological problem related to the muscular, nervous or bony function of the chest and/or abdomen.

This chapter will provide an overview of traumatic/crush/compression asphyxia, ending with the consideration of ‘burking’, a process where two different forms of mechanical asphyxia are combined for the purpose of killing an individual and selling the corpse for financial gain. Although there are mechanistic similarities, this chapter does not consider so-called ‘positional asphyxia’ as this is covered in Chapter 23.

## Ventilation

Ventilation (i.e. breathing) is accomplished through pressure changes created within the lungs. It comprises two phases. Inspiration is an active process when the diaphragm contracts, moving downwards towards the abdominal cavity, and, at the same time, contraction of the intercostal muscles causes the ribcage to move upwards and outwards. These two processes result in the net increase in volume within the chest and a reduction in pressure within the lungs. This pressure difference between the higher external atmospheric pressure and the lower intrathoracic pressure causes air (containing oxygen) to be drawn into the lungs. Following this, exhalation is a passive phase involving the relaxation of the diaphragm and intercostal muscles which results in the passive exhalation of air from the lungs.

Remembering this basic physiological process enables one to understand the pathological process resulting in traumatic/crush/compression asphyxia. Put simply, when a force is applied to the chest and/or diaphragm of sufficient magnitude to inhibit the active phase of ventilation, ventilation can no longer occur and thus air movement into the body stops. The force applied to the chest also affects blood circulation, particularly venous return from the head and neck and from below the diaphragm, which in turn explains the pathological findings typical of this form of mechanical asphyxia.

## Terminology

Three different terms are commonly used to describe the same mechanistic process where ventilation is compromised by a force applied to the chest and/or abdomen – traumatic, crush and compression asphyxia. Although considered interchangeable, the terms are subtly different, principally in relation to the circumstances in which the patient becomes compromised. Of the three terms in use, the author is of the personal opinion that the term ‘compression’ asphyxia, although undoubtedly the term used least frequently, is actually the most appropriate as it describes the mechanism that is occurring to the chest and/or abdomen to compromise ventilation, i.e. the application of a compressive force.

### Traumatic asphyxia

Although it was not called traumatic asphyxia (TA) at the time, Ollivier is credited with providing the classical description of TA in relation to 23 persons who died in a crowd incident at the Champs de Mars on 14 June 1837 [43].

> ‘It is clear that in most cases death resulted from the cessation of the mechanical phenomenon of

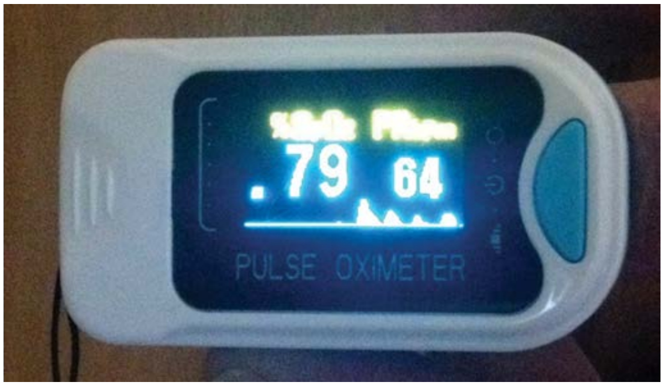

**Figure 22.1** Pulse oximeter reading of an adult male tourist visiting Puno, South America, altitude 3827 metres. He was fully alert and able to remain at this altitude throughout his visit. The oxygen saturation value reflects the reduced availability of breathable oxygen at this altitude. (Image courtesy of Dr Yvonne Littler, Leicester, UK.)

> respiration and that the violent compression of the chest walls can lead at the same time to asphyxia and brain congestion that rapidly lead to death.'
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> *Reproduced from the translated version of the original text*

Tardieu, on the other hand, maintained the view that deaths in such circumstances were related to or influenced by suffocation. Following the work of Ollivier, he wrote two narrative papers describing the pathological findings of TA in which he maintained this mechanistic view [68,69]. At the very beginning of the 20th century Perthes expanded the clinical description of TA, and it became known as 'Perthes syndrome' [46]. However, Burrell and Crandon [8] appear to be the first authors to specifically use the term 'traumatic apnoea' or 'traumatic asphyxia' in 1902. Following the publication of their paper, a steady flow of case reports emerged in the medical literature using the newly introduced and now established term 'traumatic asphyxia' [4,5,53].

## Crush asphyxia

'Crush asphyxia' (CA) is an alternative term used to describe TA. Although Burrell and Crandon make reference to the 'panic at Victoria Hall, Sunderland, June 16th, 1883, where nearly 200 children rushed into a closed corridor and were asphyxiated by crushing' [8], the first time the term 'crush asphyxia' appears to have been used within a forensic pathology textbook is in Mason's 1978 *The Pathology of Violent Injury* where Mason referred to both TA and CA in the same section, making no distinction between the two terms [41]. Unfortunately, he does not reference any early work in which the terminology was used, although in later editions [42] reference is made to Perthes' work and a 1998 review by Brinkmann [7].

Although TA and CA appear synonymous, in the post Hillsborough Stadium disaster paper of Wardrope et al. the authors make a distinction between the two terminologies [73]:

> 'We have used the term "crush asphyxia" to describe the main neurological injury in these patients. Although there were similarities to traumatic asphyxia, the conditions differ in mechanism of injury, clinical findings, main complications and outcome.'

They go on to provide the following definitions of TA and CA [73]:

> 'Traumatic asphyxia is usually caused by a heavy weight falling on the chest or a violent crush between heavy objects, such as motor vehicles. Often the victim has warning that he or she is about to be crushed, and the natural reflex is to suddenly inhale and then close the glottis. It has been suggested that this results in massive increase in pressure in the superior vena cava, explaining the classic presentation of traumatic asphyxia, when petechial haemorrhages in the superior vena caval distribution and subconjunctival haemorrhages are most always found … By contrast, crush asphyxia is caused by a gradually increasing and sustained pressure on the chest, so preventing inspiration and making it more likely that the glottis will remain open. This may lessen the increase in pressure in the superior vena cava, while the sustained nature of the crush may be important in the development of serious neurological complications.'

In their writing about death in crowds Rutty et al. [57] have expressed scepticism concerning the proposed mechanism that closing the glottis and suddenly inhaling provides an appropriate explanation for any increases in pressure in the superior vena cava. Rather, they have proposed that the increase in venous pressure and consequent development and distribution of signs of asphyxia are simply the result of transmitted back pressure effects from the chest through the superior vena cava, noting that there is inadequate protection from this occurrence due to a lack of competent valves.

## Compression asphyxia

The term 'compression asphyxia' is used to describe the mechanical limitation of the expansion of the lungs by pressure on the chest and/or abdomen. It is less frequently encountered than the other two terms within the literature and internet resources. It can be viewed as synonymous with TA and CA, as the underlying mechanism for all three is one of compression of the chest and/or abdomen. However, this compression need not be continuous as implied in the quote above, but rather – for example, as in the pressure waves experienced within crowds – may be intermittent in nature and yet still have a life-threatening effect on an individual. It is for this reason that in the most recent Hillsborough Inquiry the review pathologists favoured the term 'compression asphyxia', rather than TA or CA, to best describe the mechanistic process affecting the victims of the disaster.

## How fast do they die?

When considering death in crowds, Rutty et al. [57] draw our attention to the fact that:

> 'The process of dying is not a simple single event, but one which occurs due to the cessation of function of a number of essential organs; a process that occurs over time. This time frame is difficult to predict and comprises three different stages: the time to unconsciousness, the time to cardiorespiratory arrest and the time for the process to become irreversible (time to irreversibility). One does not inevitably follow the other.'

Considering each of these three steps in a little more detail enables us to review the paucity of information regarding the forces which may be involved in deaths related to TA, CA and compression asphyxia.

### Time to unconsciousness

To the author's knowledge, there is no peer-reviewed literature which describes the time frame in which the three forms of mechanical asphyxia may lead an individual becoming unconscious. The time frame could be a matter of seconds if the compressive forces stop all ventilatory efforts. In the book entitled *Forensic Medicine: Clinical and Pathological Aspects*, the authors of the chapter on asphyxia suggest that nerve cells in the cerebral cortex are highly susceptible to a lack of oxygen and that, as a person's natural oxygen only lasts for approximately 5–8 seconds, unconsciousness can ensue once the oxygen reserve is exhausted [18]. Such a time frame is further supported by the work of The Working Group on Human Asphyxia, Canada, who, in relation to full suspension hanging, suggest that the time to unconsciousness is only a matter of a few seconds following the application of the ligature to the neck [59]. However, as the compressive force may not be continuous but intermittent, as experienced in crowds, one must always be cautious, and not dogmatic, when it comes to suggesting time frames.

### Time to cardiorespiratory arrest

As with the time to unconsciousness, the time when a person experiencing compressive forces goes into cardiorespiratory arrest is difficult to predict, but there is a small evidence base to assist us with this consideration. The following is a quotation from Sir Thomas Smith, *De Republica Anglorum* (1583) [36]:

> '… his condemnation is to be pressed to death, which is one of the cruellest deathes that may be: he is layd upon a table, and an other uppon him, and so much weight of stones or lead laide uppon that table, while as his bodie be crushed, and his life by that violence taken from him …'

This relates to the practice of judicial 'pressing' or 'peine forte et dure', where weights were placed on the chest of an individual to extract a confession or to encourage them to plead to an indictment. It provides an insight into the amount of weight an individual may tolerate before death ensues, thus indicating that death occurred over a period of time [36] (Figure 22.2). The reports are almost exclusively of males – for example, Giles Corey who, while being pressed to death in 1692 during the Salem Witch Trials, is alleged to have asked for 'more weight'; the Catholic martyr Margaret Clitherow is the only recorded female to be pressed to death. Occurring in 1583, it is recorded that 'a quantity of seven or eight hundredweight at least' was placed on her and that she died in 'one quarter of an hour' [74]. Data available from these judicial pressings reveal that masses of up to 182 kg were survivable and over 284 kg were fatal over time [36]. This provides a lower end of the weight tolerable; fatal cases of chest compression are usually associated with becoming trapped under vehicles where masses are in excess of 1000 kg [36]. Instantaneous arrest has been reported when a weight of 907 kg was taken on top of a person [74]. Experimental work in Japan using an animal model for TA has shown that, depending on the weight applied to the chest, arrest may occur within 5 minutes 15 seconds [24]. Work referenced from the UK Home Office suggests death may occur within 15 seconds when a load of 6227 N is applied to the chest and 4–6 minutes when a load of 1112 N is applied [30]. In a recent attempt to produce a biomechanical model for the forces required to cause a flail chest, which is frequently seen in compression asphyxia deaths, Kroll et al. [36] suggest a static force of

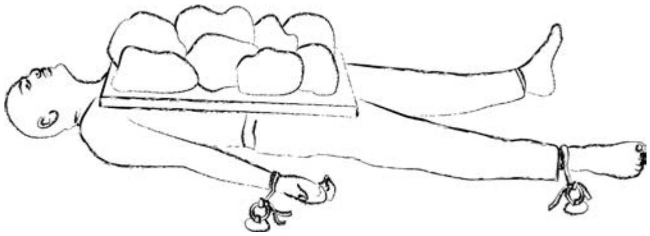

**Figure 22.2** An artist's impression of 'peine forte et dure'. A board is placed on the restrained prisoner's chest and weights are added which will have the effect of restricting ventilation. (Illustration courtesy of Vicky Eves, UK.)

over 2550 ± 250 N (i.e. similar to that observed for judicial pressings or a dynamic application of force of more than 3500 N) is required to produce a flail chest.

### Time to irreversibility

Although standard medical teaching would have us believe that irreversible brain injury may occur within 3 minutes of cardiorespiratory arrest, this may not necessarily be the case; if it were, attempts at resuscitation beyond this time frame would be pointless. Brain function will cease shortly after cardiorespiratory arrest finally occurs and, if cardiorespiratory resuscitation is not initiated or is unsuccessful, will become irreversible. The time for this to happen (time to irreversibility) is yet again not a simple matter to predict. It may also be hampered by the need to rescue an individual from beneath the compressive force, which may lead to an extended extraction time and thus a prolonged period of arrest.

## Incidents

This form of mechanical asphyxia is typically associated with three types of incidents: Deaths in crowds, and vehicle- and workplace-related incidents. Other scenarios exist.

Ollivier's original description of TA related to the victims of a crowd incident. There is probably not a year that goes by when injuries or deaths do not occur at events involving crowds. The internet resource of Still provides a review into incidents that have occurred between 1902 and the present [66]. Although TA, CA and compression asphyxia are associated with such incidents, Rutty et al. [57] point out that other forms of asphyxia and trauma may occur to the victims of such events which should be remembered by those involved in the incident investigation.

The classic papers of Perthes, and Beach and Cobb from around the turn of the 20th century illustrated vehicle- and workplace-related incidents [4,46]. Such incidents will be the commonest incident type encountered today when, for example, motorcyclists or pedestrians become trapped under vehicles or buildings collapse on top of individuals in natural disasters or workplace- or terrorist-related events. For building their biomechanical model of flail chest, Kroll et al. [36] used the example of soda vending machines falling onto people and causing their death, which illustrates the diversity of scenarios associated with this form of mechanical asphyxia.

## Pathological features

There is an abundance of publications describing the spectrum of external and internal changes that can occur in those who are subjected to TA, CA or compression asphyxia [2,3,6,10,11,13,15,17,19–23,25–29,31,32,34,35,37–39, 44,45,48–52,54,58,60–63,65,71,72]. In those dying of crowd-related incidents, the external findings typically range from a complete absence of asphyxial features to florid changes. This observation further supports the warning from Rutty et al. [57] that it would be wrong to assume that all victims die from TA, CA or compression asphyxia in such incidents.

### External findings

As is often the case in the field of forensic pathology, if one takes the time to read the historical literature, which may not be in the English language, one will come across classical descriptions of pathological processes that one still encounters today in one's own practice. The classic external findings in a traumatic/crush/compression asphyxia-related death are craniocervical congestion/cyanosis, petechiae of the head and neck, and subconjunctival haemorrhage (Figure 22.3). These were described in the victims of the Champs de Mars incident by Ollivier in his 1837 monograph [43]:

> 'In all cases, without exception, the skin on the face, neck and in some cases the upper chest, was of a uniform purple colour with multiple small bruises, blackish in colour, the widest of which were one and a half lines [lignes] in diameter, whereas the majority of them were similar in fine specks. The ocular and eyelid conjunctiva were similar affected. This remarkable colouration of the skin on the face and neck varied in intensity from individual to individual but with the same basic characteristics. The rest of the body was colourless and extremely pale.'
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> *Reproduced from the translated version of the original text*

In addition, he described blood seepage from the ocular conjunctiva, serosanguinous foam from the nose and mouth and bleeding from the nostrils and ears [43], which can occur in such cases. Perthes went on to describe other features commonly encountered in such cases, including swelling/oedema of the eyes and face, petechial haemorrhages of the buccal mucosae, soft palate and pharynx and, in his female victim, petechiae to the labial mucosae [46]. The classical so-called 'masque ecchymotique' or 'death mask' caused by the florid petechiae seen to the face and neck with a clear cut-off line between normal and abnormal skin was first illustrated to the author's knowledge in the 1904 paper of Beach and Cobb [4] (Figure 22.4).

#### Mechanistic hypotheses for the external findings

As the force is applied to the chest, intrathoracic pressure increases which in turn compromises venous return to the heart from the head and neck via the superior vena cava and the subdiaphragmatic regions via the inferior vena

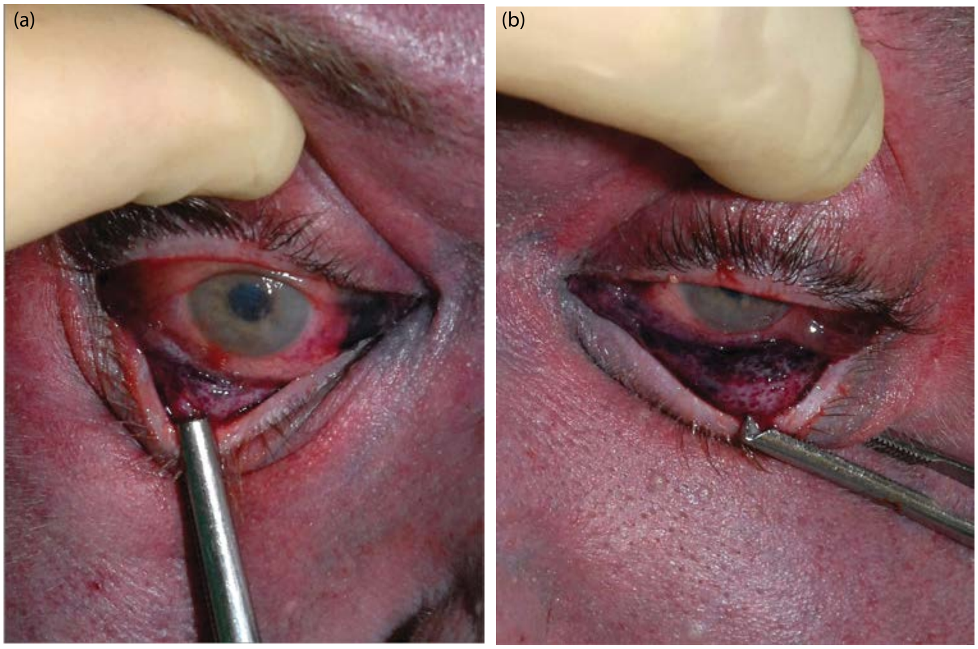

**Figure 22.3** (a),(b) Marked congestion of the face and conjunctiva with numerous petechial haemorrhages of the mucosal surfaces of the eyelids in a case of crush asphyxia.

cava. At the same time as this is happening, while the heart still beats, blood will ascend into the head and neck and descend into the abdomen where, due to the venous return being compromised, it accumulates. As it is hypothesized that the jugular veins are valveless, raised intrathoracic pressure backflow from the chest can also occur up the veins of the neck. The head, neck and, to a certain extent, the abdominal contents now become congested through a combination of arterial and reverse venous flow. Whether the resulting petechiae are attributable solely to congestion of the venules and capillaries or due to perivascular blood accumulations occurring within the unsupported structures, such as the mucous membranes, has been debated within the literature since the publication of Beach and Cobb.

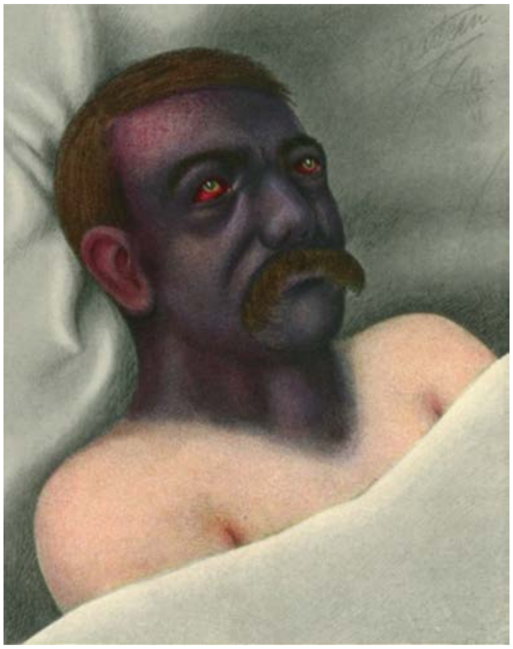

**Figure 22.4** The original image of the so-called 'masque ecchymotique' from the 1904 Beach and Cobb publication concerning traumatic asphyxia [4]. (Reproduced with permission from Lippincott-Raven.)

### Internal findings

A number of non-specific changes are reported to occur to the internal organs of the head, neck, chest and abdomen which can be explained principally due to the marked vascular congestion that occurs in these cases. These include scalp petechiae, cerebral congestion, meningeal petechiae and intracranial bleeds in the form of subdural haematoma and subarachnoid haemorrhage. Laryngeal congestion and petechiae as well as epiglottic petechiae may occur, as may oedema of the tongue. Blood may be present in the airways, with pulmonary oedema and haemorrhage observed. Thymic, cardiac and pleural petechiae may be seen, with the left ventricle on occasion being described as 'empty'. The subdiaphragmatic organs can show generalized congestion, with intestinal haemorrhage and, in living survivors, haematuria all been reported within the literature.

## Burking

'Burking' is an example of homicide where two forms of asphyxia are combined into a single act, usually smothering and traumatic/compression asphyxia. In legal terms, it means to murder an individual, usually by smothering, for the purpose of selling the corpse. It takes its name from William Burke, a 19th-century murderer who, with his accomplice William Hare, killed 16 people between 1827 and 1828 for the purpose of selling their bodies to Dr Robert Knox, a private anatomy lecturer, for use at the medical schools of Edinburgh [1,16,70]. Burke was hanged on 28 January 1829, and ironically subjected to partial dissection the next day as part of an anatomy lecture. His body was then quartered and preserved for anatomical display. As a consequence of their acts, the Anatomy Act was passed in 1832, which made body donation for the purpose of anatomical dissection possible.

Prior to Burke and Hare, so-called 'resurrectionists' or grave robbers/body snatchers had plied their trade to supply the medical school anatomists of the time with cadavers to dissect. Burke and Hare started out as resurrectionists, selling the body of an old pensioner called 'Donald' who died at Hare's house in Tanner's Close, West Port, Edinburgh, around Christmas 1827 from what was described as 'natural enough in the locality; drink and neglect', to pay off Hare's debt to his landlord [40]. Following the successful sale of the body to Dr Knox, they began their murder spree, starting with the murder of Abigail Simpson and ending with the murder of Mary Campbell (or Docherty – surname uncertain), both elderly females. Most of their victims were, in fact, female.

Nine of the victims of Burke and Hare were killed at Hare's house, another four at John Broggan's, two at Hare's stable and one in Burke's brother's house in Canongate. The victims were invited to the house where they were 'reduced to unconsciousness' with alcohol, usually whisky. From the text of the book by William Roughead, who details each of the murders and provides extracts from the trial and confessions of Burke [56], several different methods based around a theme can be identified as having been used to kill their victims. To murder Simpson:

> 'Hare clapped his hand on her mouth and nose, and the declarant [Burke] laid himself across her.'

The second victim, Joseph the miller, reportedly a very ill man, was killed in a slightly different manner in that Burke laid a small pillow over Joseph's mouth and Hare lay across the body to keep the victim's arms and legs down. To kill another victim, Burke:

> 'got astride-legs on top of the woman on the floor, and she cried out a little, and he kept in her breath … He pressed down her head with his breast … He put one hand under the nose and the other under her chin, under her mouth …'.

Finally, in the case of Mary Campbell, Hare held the victim's mouth and nose, and pressed her throat while Burke assisted him (Figures 22.5 and 22.6). In the book of Kerr [33], it is reported that Hare also pulled at least one victim around the room by the feet, although no further details about this suggestion are provided in this or the more detailed texts of Roughead [56] and MacGregor [40].

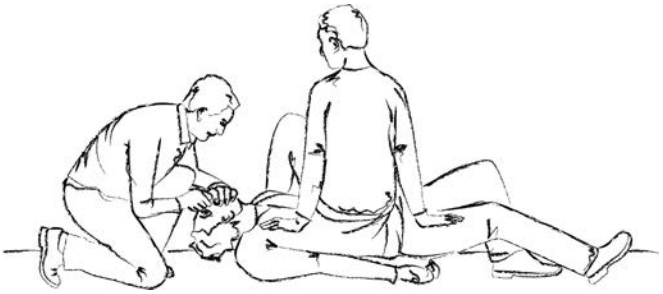

**Figure 22.5** Burking. The classical description describes the first assailant sitting, lying across or kneeling on the chest or abdomen of the unconscious, intoxicated victim while a second assailant places their hands over the nose and mouth. (Illustration courtesy of Vicky Eves, UK.)

Burke and Hare favoured the combined method they are attributed with inventing because it apparently left few signs on the victim, which in turn made the corpses more saleable to the medical schools [47,64]. The enquiry into Dr Knox concluded that [40]:

> 'These bodies do not appear in any instances to have borne external marks by which it could have been known, whether they had died by violence, or suddenly from natural causes, or from disease of short duration; and the mode of protracted anatomical dissection practised in this and other similar establishments, is such as would have made it very difficult to ascertain the cause of death, even if special inquiry had been instituted with that intention.'

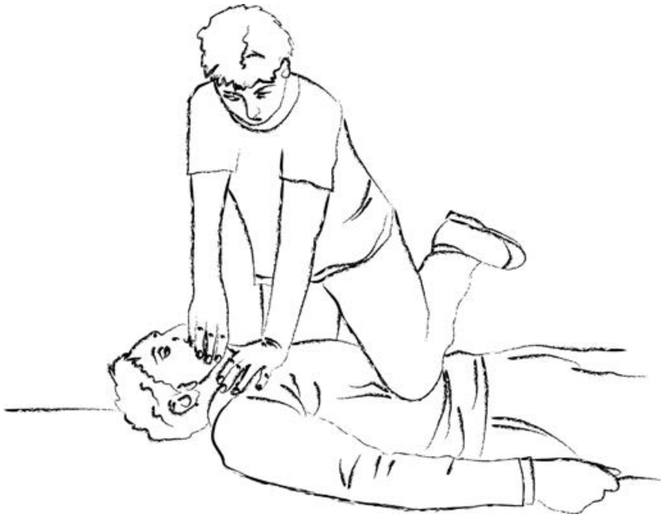

**Figure 22.6** A variation of burking with only one assailant. The assailant sits or kneels on the chest and/or abdomen of the victim while placing their hands over the nose and mouth. A hand may also be placed on the neck of the victim. (Illustration courtesy of Vicky Eves, UK.)

However, the transcript of the trial shows that the evidence-in-chief of Alexander Black, a surgeon, indicated that there were signs of asphyxia to Mary Campbell but that he was cautious in the interpretation of the signs present. Black describes seeing that blood had come from the nose, the face and eyes were swollen, and the face had a blackish hue. On giving evidence, Black stated [56]:

> 'My own private opinion was that she had died by violence; but medically, I could give no opinion, quite certain, of the cause of death'. He went on to say:
>
> 'I beg to observe that in many cases it is very difficult to form opinion with regard to suffocation; and that I really and truly believe, still, in a medical point of view, that it is dangerous to hazard that opinion.'

With regard to the postmortem examination of one of the victims, the following quotation is attributed to Professor Christison [14]:

> 'The body presented the signs of death by asphyxia – vague enough in general, and in this instance particularly so, because the method of the murderers left no external local marks … and there were no external marks about the neck or face to indicate how respiration had been obstructed.'

However, in the trial transcript presented in Roughead's book, Professor Robert Christison remarks that the external surface of the body that he examined showed several contusions on both legs, one to the left loin, a larger one to the left shoulder blade, a smaller one inside the upper lip and finally two on the head. There was dark lividity to the lips, general redness and vascularity to the whites of the eyes and ruffling of the scarf skin under the chin. Ligaments were torn within the neck, and there was a small trace of blood to the left cheek. No natural disease was observed. He discusses that he suspected that this was a case of throttling.

Possibly key to the relative absence of marks to the bodies of the victim of Burke and Hare is the fact that their victims were intoxicated to the point of unconsciousness prior to the act of burking. Thus, with the exception of so-called 'Daft Jamie', they put up little if any struggle against their assailants.

When examining a body suspected of burking, bruising may be seen on the chest and/or abdomen from the assailant's weight on the victim. The author has seen bilateral bruising to the front of the shoulder areas caused by an assailant sitting on the chest of the victim with the knees in contact with the shoulder areas. Even in the unconscious victim, bruising and laceration may be seen to the lips and gums where they are pressed against the teeth. Where the lips are pressed against the teeth, one may get bruising within the substance of the lips. Although technically challenging in terms of the reconstruction of the cadaver, Wills and Johnson describe a histological means by which such subtle bruising may be identified [75] (Figure 22.7). Again, the author has examined a case of an edentulous elderly female who was smothered by her husband. She lacked the conventional signs of asphyxia; the only marks visible to indicate an unnatural death were two tiny bruises to the inside of the upper lip where the lip had been pressed against irregularities along the gum line caused by the historical loss of teeth (Figure 22.8). Abrasion and bruising may occur to the anterior compartment of the neck although it is well recognized that the manual application of force to the neck can leave no external or internal marks. Fingernail marks from the victim and/or the assailant may be seen to the face or neck areas of the victim [12]. The assailant themselves may bear injuries caused by the victim struggling against them (Figure 22.9).

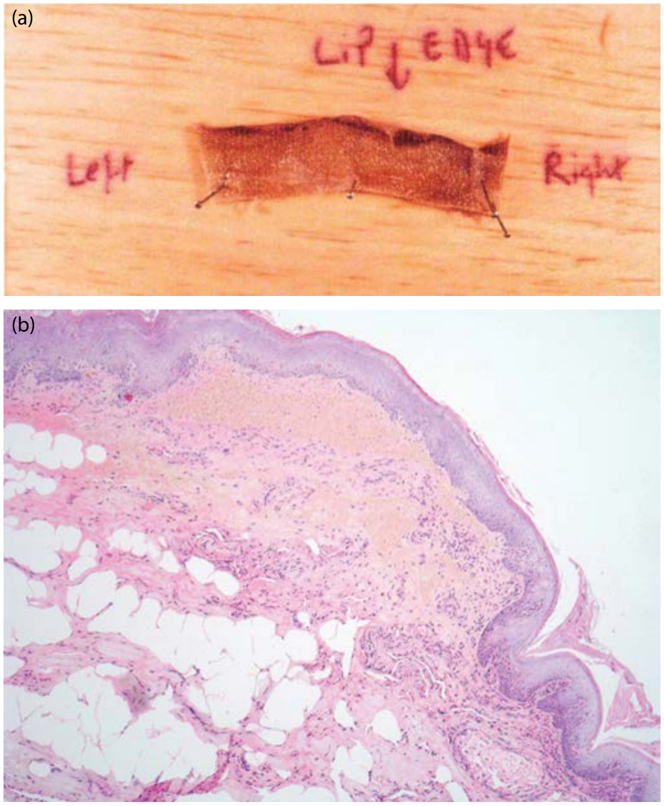

**Figure 22.7** Subtle bruising to the lips can be identified in cases of smothering. (a) The lip is first removed and orientated. (b) Histological examination identifies bruising. (Images courtesy of Dr CP Johnson, Consultant Home Office Pathologist, Liverpool, UK.)

Among other cases of so-called 'burking' described in the literature is the case of Bishop, Williams and May, convicted at the Old Bailey, London, in 1831 for the murder of a young male, the body of whom they tried to sell to the dissecting room of King's College London. The text suggests that the man came to a violent death, although the means by which this was achieved is not provided other than to describe some blunt trauma injuries to the neck and discuss how a bradawl was used to remove some of the victim's

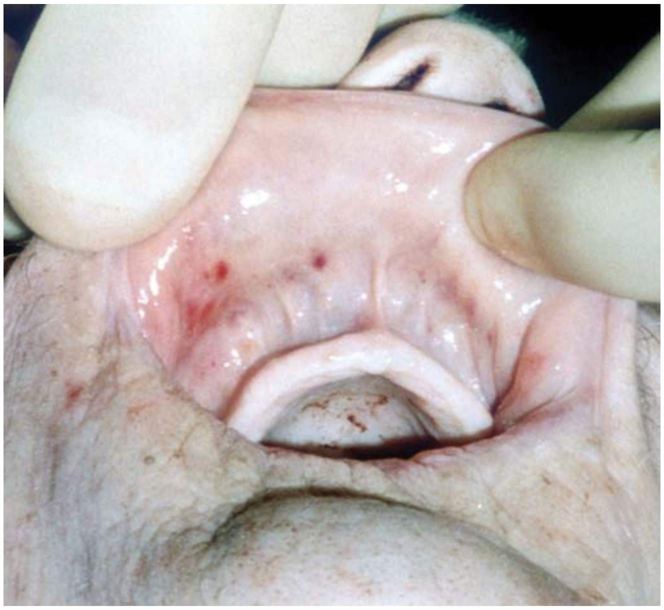

**Figure 22.8** Two bruises to the mucosa of the upper lip are seen in a case of homicidal smothering where the lip has been forced against the irregularities of the gum caused by historical tooth loss. No other external signs of asphyxia were seen in this case.

teeth [40]. In more recent times Buschmann et al. [9] have described the survivor of an assault who was subjected to traumatic and mechanical asphyxia, questioning whether the case was one of so-called burking depending upon the definition applied.

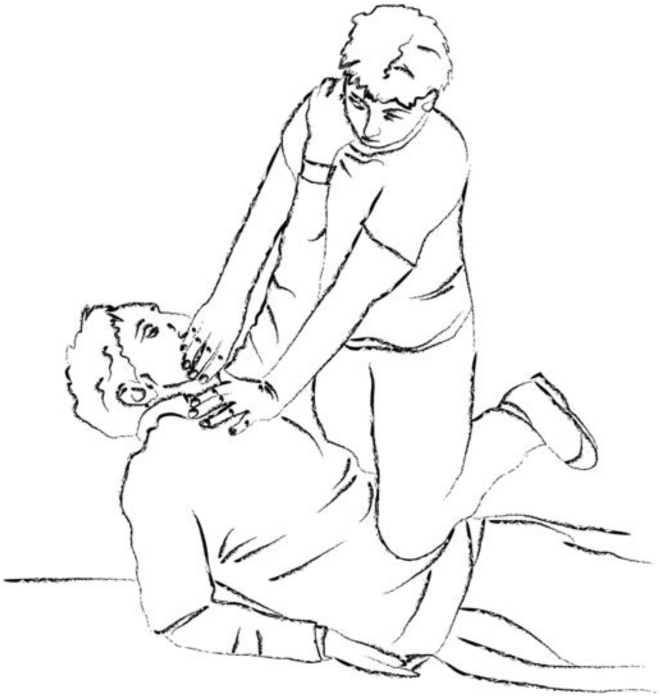

**Figure 22.9** Burking. The victim may cause injuries to the assailant, in this illustration injuries to the neck, as they struggle against the assailant. (Illustration courtesy of Vicky Eves, UK.)

## Summary

Today's forensic and legal medical practitioners will encounter single and, quite possibly in relation to crowds, multiple fatalities caused by TA, CA or compression asphyxia, three terms used to describe a common mechanistic cause of cessation of ventilation due to forcible compression of the chest and/or abdomen. Although all three terms are found in the literature and used in day-to-day practice, the most appropriate term, in the author's opinion, is 'compression asphyxia', as this accurately describes the mechanism involved. The internal changes in these cases are non-specific, however the presence of the so-called 'masque ecchymotique' or 'death mask' is typical of the florid facial features that may be seen in both the survivors of and those killed by this form of mechanical asphyxia. Few if any will encounter a case of burking when the legal definition is used to define the case, although that is not to say that the mechanism to cause the death used by Burke and Hare in the 1820s will not be encountered in modern forensic practice. If we are to believe the historical accounts of the day that burking left minimal if any marks, then if someone were to replicate the method today the case may be missed by even the best of external or more modern postmortem computed tomography-only examinations.

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# 23 Positional Traumatic and Restraint Asphyxia

*Peter Schmidt and Burkhard Madea*

## Positional asphyxia

Positional asphyxia may be defined as fatal ventilatory and cardiocirculatory failure caused by fixation of the body in an abnormal position which mechanically interferes with pulmonary ventilation by airway obstruction or interference with the inspiratory chest wall/diaphragm excursions and impairs the venous reflow to the right ventricle [19].

The following criteria for the diagnosis of positional asphyxia in cases of accidental asphyxia deaths have been suggested [1]:

- Discovery in an unusual body position interfering with ventilatory function.
- Evidence that the deceased person (decedent) placed himself/herself in the position inadvertently, i.e. without the action of another person.
- Reasonable explanation why the decedent could not escape the fatal position.
- Exclusion of alternative causes of death such as external airway obstruction, aspiration or intoxication.
- Pre-existing medical conditions not lethal on their own but probably predisposing to positional asphyxia.

Certain conditions and circumstances predispose to positional asphyxia in adults and could explain the victim's failure to free themselves. These include intoxication/sedation, accidents, organic diseases, obesity, neurological impairment/disease (e.g. multiple sclerosis, epilepsy, Parkinson's disease), loss of consciousness, physical impairment, chronic injuries, physical restraint, or combinations of the above [1,6].

Various mechanisms inducing positional asphyxia have been described [1–4]:

- Wedging of the body in a confined space (preventing movement of the chest wall and diaphragm).
- Head-down position (reverse suspension, upside down).
- Neck or trunk hyperflexion while in a head-down position or sitting upright, respectively, causing hyperextension of the neck (leading to obstruction of the upper respiratory tract).
- Flexion of the trunk over an object (e.g. the edge of a bathtub), restricting diaphragm and chest movement.
- Recreational abdominal suspension and compression.

In terms of the underlying pathophysiological mechanism [21], the fatal outcome is mainly attributed to the impairment of the ventilatory function. The failure of the inspiratory movements of the inspiratory muscles and the diaphragm may result from insufficient brain-stem motivation (e.g. from alcohol, intoxication) and respiratory fatigue caused by increased energy demand in difficult breathing as opposed to increasing hypoxia and hypercapnia [5]. Hyperflexion or hyperextension of the neck is followed by obstruction of the upper respiratory tract. Hyperflexion of the upper torso restricts the diaphragm and chest movement and diminishes the chest volume by forcing the viscera cephalad and exerting pressure on the diaphragm. In addition, the venous return to the right ventricle is reduced by the diminished negative intrathoracic pressure associated with the restricted ventilatory movements.

An analogous mechanism is also of major importance in cases of reverse suspension (head-down position) [10,20]. These are found mainly in sports accidents (e.g. alpinists, speleologists, parachutists) but also occur in accidents associated with certain sexual activities. These scenarios have in common that hydrostatic pressure increases in the upper part of the body, which does not have at its disposal compensatory mechanisms like the venous valves of the lower body. Increased hydrostatic pressure causes pooling of blood in parts of the upper body and decreased flow of venous blood to the right ventricle, followed by hypovolaemic shock. On the other hand, in crucifixions, death may be attributed to a combination of dehydration, hypovolaemic shock, and positional or mechanical asphyxia, because the chest cage and intercostals are fixed in a maximal inspiratory position when the crucified individual is raised up (see also Chapter 24).

In every case of positional asphyxia, physical stress/panic-induced catecholamine release with subsequent arrhythmias may also contribute to the occurrence of death. These effects may be aggravated by the alcoholization frequently found in the victims of positional asphyxia [15]. Alcohol was shown to decrease total peripheral resistance, arterial blood pressure, heart rate and myocardial contractility. Furthermore, an increased endogenous catecholamine response and (supra)ventricular arrhythmias were observed. At the least, alcohol may depress the respiratory centre in the brain stem and relax the muscles, in particular the genioglossal muscle which draws the tongue forward during inspiration and prevents its lapse into the pharynx [1]. According to Schoenmackers' concept of death due to abnormal stress exposure ('critical stress') [22], death occurs if the capacity of the organism is overburdened (i.e. the individual capacity limit is exceeded). If the organism is already impaired by other independent pre-existing factors, the primary stress level is elevated to a secondary stress level. As a consequence, a comparably lower additional stress exposure exceeds the capacity limit and causes the death of the individual. In positional asphyxia, the primary stress level of a healthy individual may be markedly elevated by pre-existing medical conditions, alcohol or drugs, and positional asphyxia is the final crucial impact exceeding the critical capacity limit [15,21].

The morphological findings [21] are relatively scarce and unspecific. On external examination, a peculiar distribution of livor mortis and varying patterns of minor injuries, such as scratches, haematomas and abrasions corresponding to the scene of discovery, may be found, as may petechial bleedings of the skin and conjunctivae, cephalic swelling and congestion. On internal examination, congestion of the base of the tongue, epiglottis and trachea may be encountered. Congestion and oedema of the brain and the lungs are observed, with the mean combined lung weight amounting to more than 1000 g. Haemorrhages in the chest and neck muscles indicate rupture of muscular fascia resulting from strained respiratory movement. Haemorrhages in the muscles of the axillae and upper back, in the pectoralis muscles, as well as in the periosteum of the clavicle at the junction with the sternocleidomastoid muscle, are attributed to the convulsive phase of asphyxiation. In cases with abdominal suspension, bruising of the chest wall, diaphragm, peripancreatic area and spleen may be found. In addition, underlying organic diseases may be identified which are not lethal on their own but may have led to a collapse or loss of consciousness resulting in the asphyctic position or the incapacity of the victim to free themselves.

## Case report

A 23-year-old male was found dead at 6:00 a.m., in a grotesquely fixed prone body position in the corner of a staircase (Figures 23.1 and 23.2). The decedent's left leg was outstretched adjacent to the stair rail, and the right leg was inflected and wedged on another stair rail. The left hand was found on the corner wall in 90-degree retroflection, whereas the cyanotic right hand was hanging down loosely. The decedent's cyanotic head was retroflected in a maximum position, and the nose and lips were pressed against the wall. There were no signs of self-liberation efforts, such as broken finger nails. A reddish perpendicular mark was found on the wall, beginning approximately 20 cm above the mouth.

Investigations revealed that the young man had been out the evening before and had consumed excessive alcohol within a short period of time. Subsequently, he had felt sick and vomited repeatedly. Friends took him home, where they watched him independently unlocking the door and entering the house. The next morning, he was found dead as described above. The decedent had been healthy and in good physical and mental condition prior to his death;

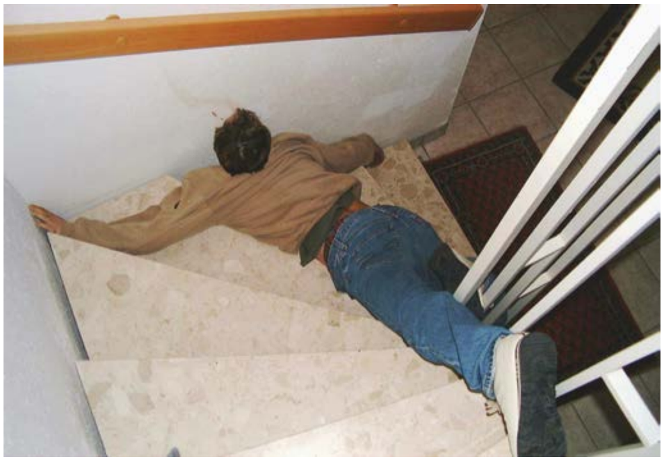

**Figure 23.1** Scene, top view. A young man ended up in a fixed prone body position after falling down the staircase under marked influence of alcohol.

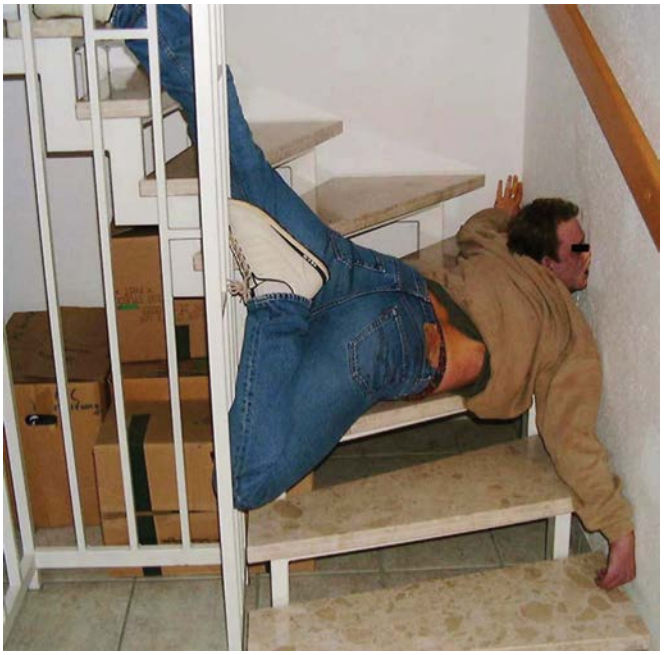

**Figure 23.2** Scene, side view. Note the maximum retroflection of the cervical and lumbar spine. There is marked cyanosis of the head and livores of the right hand. A small trace of reddish saliva can be seen on the wall above the nose. The position of the hands gives no evidence for unsuccessful self-liberation attempts prior to death.

there was no pre-existing medical history, such as cardiac or pulmonary disease or epilepsy.

## Autopsy findings

On external examination, the body showed marked petechial haemorrhages of the conjunctivae and the oral mucosa, with large-area impact abrasions and perpendicular parallel scratch marks on the left cheek and zygomatic region respectively. In the forehead skin, 3 cm above the left eyebrow, numerous subtle reddish-brown abrasions were observed in a 3 cm × 3 cm area. There was a 1.5 cm (vertical) and 0.5 cm (horizontal) reddish-brown impact abrasion on the external margin of the left palpebral fissure. In the skin between the left external auditory canal and the mandible, a large-area reddish-brown abrasion with numerous perpendicular parallel scratch marks was noted. From the lower margin of this area, eight blood streaks, up to 3 mm wide, were noted; the longest of them reached the left clavicle (Figure 23.3a). A 1.5 cm (horizontal) and up to 8 mm (vertical) streaky abrasion was observed on the margin of the left nostril, and there was a 1.5 cm (horizontal) and up to 7 mm (vertical) laceration of the left lower lip close to the corner of the mouth, bordered partially with traces of white colour. Small particles of white colour were also found in this laceration. A 3 cm (horizontal) and 1.5 cm (vertical) superficial abrasion was observed above the left mandible, again with white colour deposits (Figure 23.3a). The right side of the face and skull did not reveal any injuries. Lacerations of both knees were found. Injuries such as broken fingernails which could have been caused by self-liberation attempts were not noticed. Rigor mortis was fully developed. The distribution of livores was consistent with the position of the body at the scene.

Autopsy revealed haemorrhages of the prevertebral cervical musculature (Figure 23.3b) but no signs of external force against the neck. Furthermore, Simon’s bleedings were found. Massive haemorrhagic pulmonary oedema (right lung 970 g, left lung 900 g) and marked cerebral oedema were disclosed. Examination of the skull did not reveal any injuries. In particular, there were no abrasions or haematomas of the scalp and the cranial bone was found intact. The meninges were without pathological findings with regard to epi- or subdural haematomas. No cerebral contusions or haemorrhages were disclosed either. The cervical spine was frozen and examined after cross-sectioning, with no fractures or haemorrhages observed; the spinal medulla was found intact. In particular, the heart and coronary arteries revealed no macroscopic abnormalities, nor were there any histological signs of inflammation or ischaemia. Microscopic investigation of the internal organs did not reveal any abnormal findings; specimens from various brain regions revealed signs of acute congestion and generalized brain oedema, but no intracerebral bleeding or contusion.

## Toxicological investigations

Blood alcohol concentration was determined to be 2.60 g/l; urine alcohol concentration was 3.26 g/l. Toxicological assays of autopsy specimens for drugs of abuse applying routine methods revealed negative results. Positional asphyxia after blunt head trauma under the influence of alcohol was determined as cause of death.

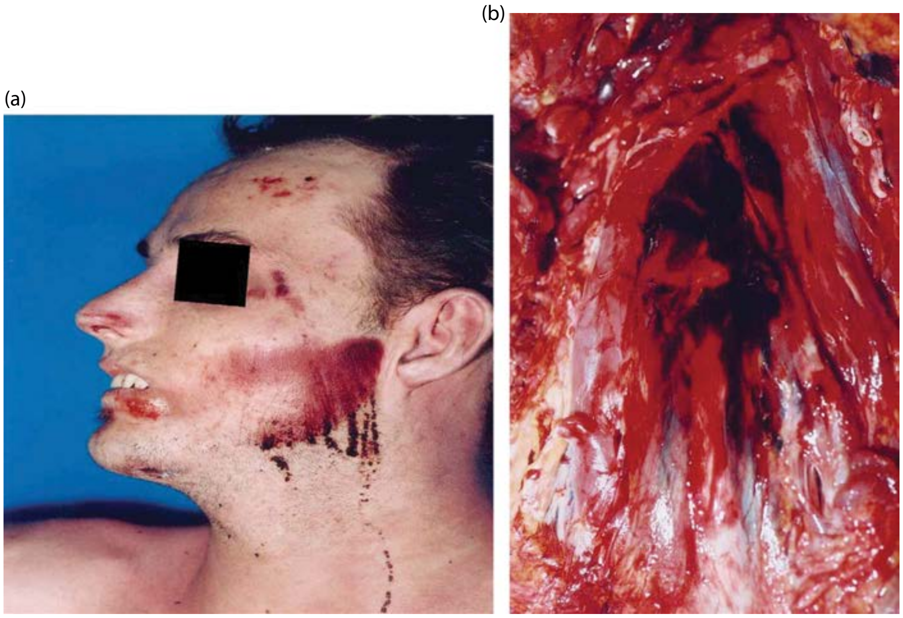

**Figure 23.3** (a) On external examination, large-area abrasions on the left cheek/zygomatic region and perpendicular parallel scratch marks were noted. Traces of white wall colour can be seen on the left lower lip and mandible, as can vertical blood streaks. (b) Praevertebral haemorrhages caused by maximum retroflection of the neck in a fixed restraint position.

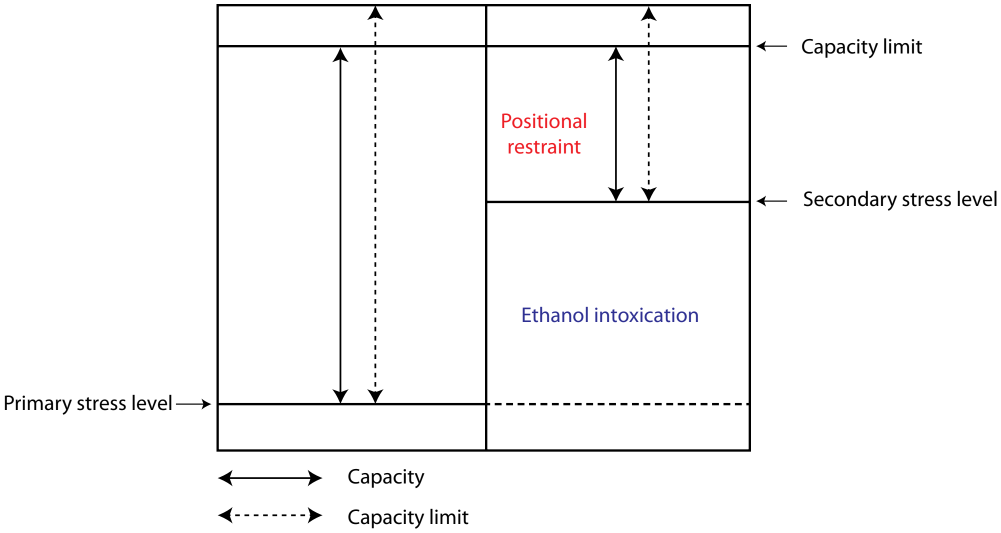

**Figure 23.4** Scheme of stress levels and capacity limit according to a concept of Schoenmackers [22].

## Conclusions

Without exception, the present case meets the criteria listed above. Thus, asphyxia due to positional restraint has to be considered as a factor contributing to the occurrence of death.

Applying Schoenmackers' concept of critical stress (described above) to the reported case, the primary stress level had been markedly elevated due to ethanol and concussion of the brain. Positional restraint (asphyxia) was the final crucial impact, exceeding the critical capacity limit (Figure 23.4).

This report demonstrates a remarkable case of positional asphyxia caused by a peculiarly fixed body position resulting from a severely inebriated young man's fall down a staircase. The findings at the scene together with the autopsy results met the criteria for positional asphyxia. Acute alcohol intoxication and concussion of the brain were determined as relevant co-factors, aggravating the impact of the positional restraint as considered co-causes of death.

## Traumatic asphyxia

The various definitions of traumatic (mechanical) asphyxia provided in the literature may be summarized as external chest or abdomen compression by a heavy object [19].

Accidental mechanisms include severe automobile crashes with wedging of the car occupants between parts of the vehicle, riot crushes in mass disasters, construction accidents or trapping of children under heavy objects. As homicidal mechanisms, crushing of a person with a heavy object and compression of the victim's chest by thighs and knees ('leg scissors') leading to 'jackknife' injuries or 'accordion' compression of a child's thorax have been reported.

The central pathophysiological mechanism is the compression of the thorax or abdomen, which results in an inhibition of the respiratory movements and a decrease of the venous reflow to the right ventricle due to a reduction of the negative intrathoracic pressure. In addition, the absence of venous valves in the inflow region of the superior vena cava allows for a reflux of venous blood, resulting in cerebral congestion and hypoxaemia. The discrepancy of weight between the compressing object and the individual involved as well as the duration of the compression mainly determine the outcome.

On external examination, typical but rather unspecific findings are encountered. These findings include a significant purple-red discolouration, oedema, petechial bleedings, subconjunctival ecchymoses and mucocutaneous bleeding of head and neck ('masque ecchymotique'). Depending on the body level of compression, the ecchymoses may also be located at other parts of the body, even the lower limbs (popliteal fossa), sparing the pressure points. The pattern of probable injuries must be consistent with the accidental or homicidal mechanism, and the severity of injuries must not be of lethal significance on their own.

The internal findings consist of internal petechiae and ecchymoses, congestion of the internal organs, in particular lungs and brain, and fractures or lacerations of internal organs, which fit together with the underlying events as concluded from the police investigations and, again, are not lethal on their own. Microscopic changes attributable to hypoxia, such as vacuolization and swelling of hepatocytes, renal tubular epithelial cells or cardiomyocytes, as well as bone marrow or fat microemboli of the lung resulting from the blunt force injuries, provide further evidence supporting the diagnostic conclusions.

Results of the toxicological investigation excluding a lethal intoxication complete the comprehensive spectrum of diagnostic forensic tools [21].

## Physical restraint

Physical restraint is discussed separately to distinguish distinctly between fatalities with the decedent being forced into the unusual body position by third parties on the one hand and positional asphyxia as a rule implying accidental entrapment of the decedent on the other hand.

This entity mainly includes the sudden death of individuals brought into a restrained prone position by law enforcement authorities to overcome resistance and to incapacitate. Generally, the wrists and ankles are restrained behind the back and bound together with a cord or hobbling device (into the so-called 'hog-tied' position), probably with additional weight on their backs compressing the thorax with the restrainer's body weight or pressure on the neck [8,11,12,16,17]. Another scenario is the subduing of patients who are acutely psychotic or in a state of excited delirium, using techniques including the 'therapeutic basket hold' in mental health clinics [23].

If the death is unequivocally attributable to an impairment of gas exchange by blockage of the nose and mouth, the term 'smothering' appears more appropriate. When compression of the neck is the main contributory factor in the occurrence of death, the fatality should accurately be ascribed as 'strangulation'.

Evidence supporting the adverse effects of the prone restraint position on ventilation and circulation was provided by a study on healthy individuals who were placed in the 'hog-tied' position after a period of exercise on a stationary cross-country ski machine to a maximum heart rate of 120 beats per minute [7]. The subjects in the restraint position exhibited a prolonged recovery time for both heart rate and peripheral oxygen saturation as measured by transcutaneous ear probe. The significance of these results was doubted because the method to determine oxygen saturation was considered inaccurate, as no direct measure of ventilatory function had been performed and the findings were assessed to be inconsistent with results from exercise physiology. Therefore, in a succeeding study [8], healthy volunteers underwent pulmonary function testing (PFT) in a sitting, supine, prone and restraint body position and in a second series of experiments were objected to a 4-minute exercise period followed by a 15-minute period in either the sitting or the restraint position. Using serial arterial blood gas measurement, electrocardiographic and pulse oximetry monitoring and PFT (including forced vital capacity, mean forced expiratory volume and maximal voluntary ventilation), a progressive restrictive pattern of PFTs was found depending on the body position (sitting to supine to prone to restraint), but exercise itself resulted in no further impairment. Conclusions from these experiments with regard to forensic-relevant conditions of restraint asphyxia are, however, limited because of significant differences in the setting. The volunteers were healthy, had a negative testing for alcohol and drugs, and had experienced no struggle and no psychological stress. Consequently, the results are not inconsistent with the concept that a combination of factors including medical condition, intoxication, agitation, delirium and struggle may predispose for a final interference of body position with breathing resulting in complex pathophysiological interactions causing the occurrence of death [8,18]. For the pathophysiological implications of alcohol, see above.

Most individuals involved exhibited behaviour consistent with excited delirium [13,14], which is characterized by constant, purposeless, often violent activity coupled with incoherent or meaningless speech and hallucinations with paranoid delusions and has a variety of causes (Table 23.1). An overview of the characteristic features of 14 restraint-related deaths and excited delirium syndrome in Ontario (2004–2011) is provided in Table 23.2 [11]. This condition on its own already predisposes to sudden death due to neurally mediated cardiac arrest.

**Table 23.1 Causes of delirium/psychosis**

| Cause | Example(s) |
|---|---|
| Drug intoxications/withdrawals/noncompliance | |
| Acute functional psychosis | Schizophrenia<br>Acute mania |
| Endocrine/metabolic disease | Hyperthyroidism, hypothyroidism ('myxoedema madness')<br>Hypoglycaemia<br>Pituitary disease with secondary endocrine effects<br>Postpartum psychosis<br>Porphyria<br>Liver and kidney failure (uraemia) |
| Electrolyte disorders | Acid–base imbalance |
| Nutritional disorders | Thiamin, vitamin B12 deficiency ('megaloblastic madness') |
| Infections | Meningitis<br>Encephalitis<br>Sepsis |
| Neoplasia (brain tumours) | |
| Seizure disorders | Complex partial seizures ('temporal lobe' or 'psychomotor' epilepsy)<br>Postictal state |
| Vascular disorders | Hypertensive encephalopathy/cerebral infarcts<br>Systemic lupus erythematosus<br>Disseminated intravascular coagulation |
| Trauma | Concussion<br>Subdural, epidural, subarachnoid haematoma |
| Hypothermia/hyperthermia | |
| Hypoxia | |
| Factitious psychosis/malingering | |

*Source:* From Gill JR. *Forensic Sci Med Pathol* 2014;**10**:223–228.

*Note:* Delirium is an acute, confusional syndrome with a transient disturbance in consciousness and cognition that has a variety of causes.

**Table 23.2 Restraint-related deaths in excited delirium in Ontario (2004–2011)**

| | Years | |
|---|---|---|
| **Detail** | **2004–2011** | **1988–1995** |
| Number | 14 | 21 |
| Mean age (SD) | 35.7 (8) | 33 (10) |
| In custody | 14 (100%) | 18 (86%) |
| Psychiatric illness | 2 (14%) | 12 (57%) |
| Drug-induced | 12[a] (86%) | 9 (43%) |
| Cocaine (lethal range) | 3/11 (27%)[b] | 0/6 |
| Violent encounter | 11/14 (79%) | 21 (100%) |
| Neck pressure | 0 | 3 (14%) |
| Prone position | 3[c] | 18 (86%) |
| Supine | 1[d] | 0 |
| Hog-tied[e] | 1 | 2 |
| Seizure preceding death | 3 (21%)[f] | 0 |
| Cardiovascular disease | 4 (29%) | 4 (21%) |

*Source:* From Michaud A. *J Forensic Leg Med* 2016;**41**:30–35

*Abbreviation:* SD, Standard deviation.

[a] Cocaine in 11 cases, methamphetamine in 1 case.

[b] No blood levels available (see limitations).

[c] No information available for 9/14 individuals; includes fatal collapse following CEW [conducted energy weapon = taser].

[d] Very combative with police officer – was put in police wagon with handcuffs and ankle restraints only – was struggling side to side in police wagon – still combative at hospital arrival – unexpected death occurred when restrained supine at hospital arrival – no detail on restraint technique.

[e] Hog-tied and left as such in prone position.

[f] 2 Cocaine-induced ExDS [excited delirium syndrome] and 1 methamphetamine-induced ExDS.

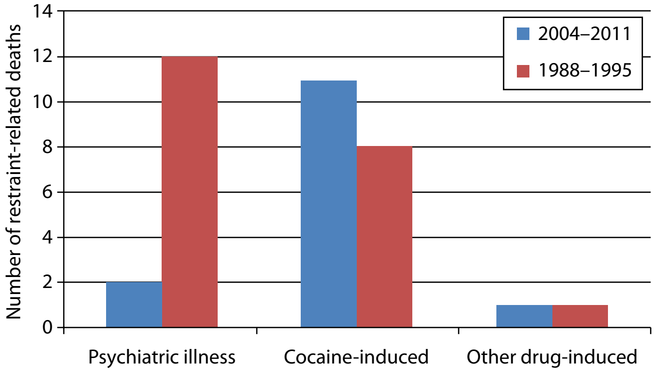

**Figure 23.5** Cause of excited delirium syndrome in restraint-related deaths in Ontario (according to Michaud [11]).

In the individuals involved, excited delirium was most often caused by a psychiatric disorder, but it was also frequently induced by psychoactive stimulative drugs, in particular cocaine (Table 23.3, Figure 23.5) [9,11,16]. Cocaine causes excessive sympathetic activation by blocking presynaptic uptake of catecholamines, dopamine and serotonin, resulting in increased myocardial contractility, blood pressure and heart rate. Coronary artery vasoconstriction, increased microvascular resistance and enhanced thrombogenicity of the blood may be associated with myocardial ischaemia [14].

**Table 23.3 Drugs associated with acute psychosis**

| **Drug group** | **Example(s)** |
|---|---|
| Ethanol | Ethanol intoxication<br>Ethanol withdrawal |
| Sympathomimetic drugs | Cocaine<br>Amphetamines<br>Tricyclic antidepressants<br>Monoamine oxidase inhibitors<br>Methylphenidates |
| Sedative drugs | Sedative drug withdrawal |
| Hallucinogens | Cathinones<br>Lysergic acid diethylamide (LSD)<br>Mescaline<br>Phencyclidine (PCP)<br>Psilocybin |
| Anticholinergic drugs | Includes drugs with anticholinergic side effects, antipsychotic agents, antihistamine, etc. |
| Corticosteroids | |

*Source:* From Gill JR. *Forensic Sci Med Pathol* 2014;**10**:223–228.

In conclusion, the mechanism of death may be a fatal cardiac dysrhythmia or respiratory arrest induced by the interaction of several factors resulting in an imbalance of increased oxygen demand and decreased oxygen delivery. Psychiatric or drug-induced stress of agitated delirium coupled with police confrontation generates catecholamine mediated stress (heart). The hyperactivity associated with excited delirium, struggle with the police and ventilatory work to overcome the restraint increases the oxygen delivery demands. Finally, the hog-tied position inhibits chest wall and diaphragmatic movements, thus impairing ventilation in a situation of high oxygen demand [13].

The diagnosis of fatal 'restraint asphyxia' is based on the following criteria:

- The circumstances surrounding death or the fixation in a hog-tied position may have interfered with respiration. (The mechanics of the restraint have to be elucidated in detail by interviews with the witnesses.) Methods used to restrain people are summarized in Table 23.4.

**Table 23.4 Methods used to restrain 21 people**

| **Method of restraint** | **Number (and %) of people** |
|---|---|
| Prone | |
| With chest compression | 4 (19) |
| With handcuffs | 4 (19) |
| With handcuffs and ankle shackles* | 6 (29) |
| With chest compression, handcuffs and ankle shackles | 4 (19) |
| Pressure applied to neck | 3 (14) |

*Source:* From Pollanen MS et al. *CMAJ* 1998;**158**:1603–1607.

* Includes 2 people who were 'hog-tied'.

**Table 23.5 Characteristics of 21 people with excited delirium who died unexpectedly after being restrained (Ontario, 1988–1995)**

| | Sex; number | | Mean age in years (and SD) | Circumstances surrounding death; number (and %) | | |
|---|---|---|---|---|---|---|
| **Cause of excited delirium** | **Men** | **Women** | | **In police custody**[*] | **Subdued with pepper spray** | **Had heart disease**[†] |
| Psychiatric illness | 12 | 0 | 35 (11) | 10 (83) | 4 (33) | 2 (17) |
| Cocaine-induced psychosis[‡] | 7 | 1 | 31(7) | 7 (88) | 0 | 2 (25) |
| Toxic effects of other drugs[§] | 1 | 0 | 17 | 1 (100) | 0 | 0 |
| **Total** | **20** | **1** | **33 (10)** | **18 (86)** | **4 (19)** | **4 (19)** |

*Source:* From Pollanen MS et al. *CMAJ* 1998;**158**:1603–1607.
*Abbreviation:* SD, Standard deviation.

[*] Either in prison or while under arrest.

[†] Hypertensive or atherosclerotic heart disease.

[‡] Includes one person with multiple drug intoxication (pseudoephedrine and dextromethorphan) and another with a history of cocaine use but no evidence of cocaine use just before death.

[§] Combined effects of alcohol, morphine, diazepam, acetaminophen and marijuana.

- Historical information on the event indicates difficulties in breathing or unusual physical respiratory signs, such as cyanosis, gurgling, gasping or any other physical manifestations that could be interpreted as evidence of respiratory distress. (Identifying the length of time under restraint involved and the coincidence of loss of consciousness and restrained position that compromises breathing is essential for establishing a cause-and-effect relation with death.)
- Pre-existing pathological conditions, in particular hypertensive or atherosclerotic heart disease, may be found, but a medical condition or an intoxication which would conclusively account for death on its own is excluded.

Characteristics of 21 people with excited delirium who died unexpectedly after being restrained are shown in Table 23.5.

To sum up, in the individual case of prone restraint asphyxia, forensic expert opinion must deal with the issue of whether the forcible restraint per se may be established as the sole cause of death or whether contributory factors such as catecholamine rush, neuroleptic malignant syndrome, psychogenic death (including exhaustive mania and excited delirium) or exercise-induced cardiac arrest have also to be taken into consideration [9].

## Conclusion

Positional asphyxia, traumatic asphyxia and physical restraint have in common that the pathophysiological effects of an unusual position of the body or an external compressive impact on the thorax or abdomen which impairs the integrity of the upper respiratory airways, the inspiratory movements of the thorax or the venous reflow to the right ventricle initiate the lethal sequelae in individuals who are predisposed by pre-existing medical conditions or impairment by alcohol or drugs which may be assessed as non-lethal on their own. Based upon a comprehensive medical examination, the diagnosis is established by exclusion considering the circumstances of death, in particular the position of the body at discovery, the minor injuries consistent with the lethal events and the unspecific findings attributable to mechanical impairment of respiratory function or venous congestion.

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# 24 Death Upside Down

*Burkhard Madea and Elke Doberentz*

## Introduction

There are a number of case reports in the literature on deaths with orthograde (feet down) suspension of the body. The cause of death in these cases was mainly due to hindering of respiratory excursion, in some cases together with orthostatic collapse. All authors reporting on these cases referred to analogies concerning the death on the cross [4,5,8,9,21,37,45,59].

In contrast, there are only a few reports on deaths in a head-down position [27,36,46,47] (Table 24.1). In some of these cases no autopsy was performed [27,46]. Based on the circumstances, the cause of death was thought to be 'cardiac arrest' or 'cardiac death' (differential diagnosis, diabetic coma), or accident with following 'orthostatic collapse'. In those cases with a complete postmortem, no clear anatomical cause of death was found [36,47]. Marshall [36] mentioned in his paper that, during the Second World War, torture had on occasions been inflicted by hanging the victim upside down by the ankles. Obviously, such tortures also happen nowadays and forensic pathologists or police medical officers may be asked for their opinion on how long it might be possible to survive in a head-down position. There seems to be little knowledge available to answer this question.

Historically, death in a head-down position was not rare, when different death penalties are taken into account. For instance, an altar painting from Masaccio (1401–1428) shows the crucifixion of St Peter in a head-down position and torturing in a head-down position is seen in a drawing by Goya. There are many further paintings on death in a head-down position. According to old reports, the agonal period after hanging in a head-down position lasts several hours, even a day [40].

Cases of death in a head-down position are reported briefly here.

## Case reports

### Case 1

An 85-year-old woman was brought to hospital suffering from vague abdominal pain. The tentative diagnosis was bowel perforation. Diagnostic radiography of the colon by means of barium enema was commenced. After the tube was inserted into the rectum and the barium enema was instilled, the woman was brought into a 30-degree oblique position with her head down. Circulatory arrest occurred in this position.

At autopsy, the main anatomical findings were calcified aortic stenosis combined with aortic insufficiency; hypertrophy and dilatation of the left ventricle and insufficiency of the mitral valve; dilatation of the left atrium; pulmonary oedema; advanced atherosclerosis; old infarction of the right kidney; and diverticulosis of the descendent colon but no bowel perforation.

The cause of death was given as left heart failure due to severe aortic stenosis. In this case the autopsy revealed a sufficient anatomical cause of death, and death occurred accidentally in a head-down position, but it could have occurred due to the severe aortic stenosis in any position.

### Case 2

A 56-year-old man was found in a head-down position, hanging in a sack. The sack was knotted to a board which was laid over a door and cupboard. Tennis balls were wrapped into the free end of the sack and these 'enveloped' tennis balls fixed the knots of the rope at the sack so they could not slip. The sack was hanging from the board (Figure 24.1).

The stitching of the 'head end' of the sack was opened for a length of 15 cm and the hands jutted out through this opening. The right hand held a pair of scissors, between the blades of which fibres of the sacking had impacted to prevent further cutting actions. The body was tightly pressed into the sack, and beside the body were two chairs, one overturned. The man had last been seen 2 days earlier.

#### Autopsy findings

The man was covered only with a blue plastic sack. There was deep hypostasis of the head, face and arms as well as the internal organs of the head, neck and thorax. Cuff-like bleeding into the subcutaneous fat of the arms corresponded to the grooves where the arms passed through the sack. There was brain and lung oedema, but no anatomical cause of death. Toxicology was completely negative.

**Table 24.1 Summary of the published cases of death from reverse suspension**

| Author(s) | Case report |
|---|---|
| Prokop and Radam [46] | Elderly woman found dead in a head-down position hanging out of a window with the left foot fixed between the window and the window frame.<br>No autopsy. Differential diagnosis: diabetic coma, cardiac death, accident with orthostatic collapse. It remains unclear whether the woman died in this position or came dying into the head-down position. |
| Hilgermann and Richter [27] | 72-year-old man. Accidental death during autoerotic practice simulating his own slaughter. The man had simulated a situation comparable to the slaughter of a pig.<br>No autopsy. Cause of death was thought to be circulation arrest in a head-down position. |
| Marshall [36] | 11-month-old child found dead in a head-down position, 4 hours after last having been seen alive.<br>Autopsy revealed no anatomical cause of death. |
| Marshall [36] | 60-year-old man found dead about 10.5 hours after last seen alive, suspended by his clothing upside down in a thorn hedge. It seemed that, in his intoxicated state, he had tripped and fallen head first over the hedge.<br>BAC: 230 mg/100 ml.<br>Autopsy revealed no clear anatomical cause of death. |
| Purdue [47] | 48-year-old man found suspended upside down from a security fence with the right foot jammed sideways between two adjacent spikes of the fence.<br>BAC: 129 mg/100 ml.<br>Autopsy revealed no clear anatomical cause of death. |
| OG Williams [pers. comm.] | Middle-aged man, mental disturbance, found after several weeks' absence in a derelict house. Floorboards upstairs were partly missing, exposing the wooden beams which support the floor. Found dead hanging upside down with the backs of the knees across a beam and his feet hooked under the next beam: all of the body from the knees downwards was hanging vertically.<br>Advanced decomposition. No pathological findings to account for death. |
| B Knight [pers. comm.] | Thief trying to get into a house through the fanlight. Age about 20–30. Found dead with the upper part of the body from the hips hanging down inside and his legs hanging down outside.<br>BAC: negligible<br>No anatomical cause of death. |
| B Knight [pers. comm.] | 40-year-old man, tried to climb into his friend's house. He stood on the outside lower window ledge, but scratch marks on this indicated that his feet had slipped off and left him hanging partly inside the fanlight with his head and arms hanging downwards.<br>BAC: 110 mg/100 ml. |
| Lawler [32] | 62-year-old derelict found dead with his right food trapped beneath part of a broken board and with his body suspended. BAC and toxicology negative. Accident occurred in cold January. Several gastric erosions. |

*Source:* Adapted from Madea B. *Forensic Sci Int*. 1993;**61**:119–132.

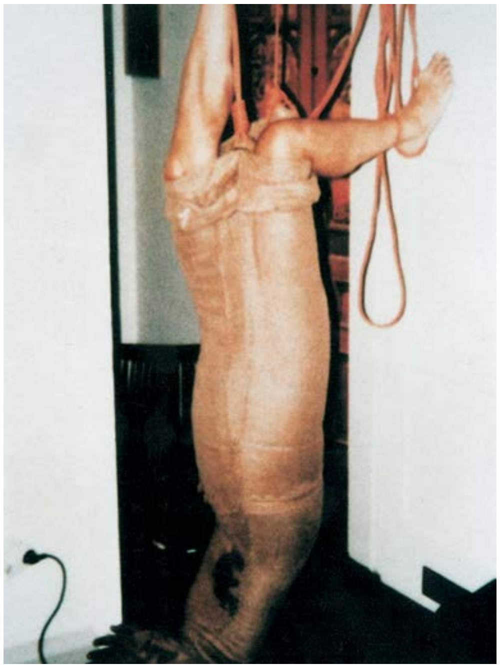


**Figure 24.1** The deceased at the scene.

## Histology

The findings were severe congestion of the organs of the upper part of the body, especially the lungs; vacuolization of liver cells due to hypoxia; and cuff-like haemorrhages of the subcutaneous fat of the forearms with marked infiltration of polymorphonuclear leucocytes. The whole case was interpreted as an autoerotic death.

Obviously, the man – standing on the chairs – covered his body with the sack and used the tennis balls to compress his genitals. One of the chairs must have fallen over and the man came into a head-down position due to the fact that the turning point was below the centre of gravity of the body.

It was surmised that he had taken scissors into the sack to rescue himself in case of accident, but he was able to cut only the stitching of the head end of the sack. In this attempt at self-rescue, the man had inflicted several sharp cuts to his forehead.

According to the morphological findings (emigration of polymorphonuclear leucocytes and signs of general hypoxia) and the circumstances of the case (attempted self-rescue), it was clear that the man had been alive and conscious in the head-down position and died in this position. However, the autopsy revealed no clear morphological cause of death.

## Further case reports

We have knowledge of several other similar cases through personal communications. Professor Bernard Knight of the Wales Institute of Forensic Medicine has reports of two deaths where men became impacted while attempting to climb through a high, narrow window, so that they were 'jack-knifed' across the hips, with the head, arms and upper body hanging down inside. Both showed only gross facial congestion, cyanosis and some conjunctival petechiae. Two other drunken persons were found having partially fallen out of bed, with their heads on the floor and legs still in the bed. Both had gross facial ecchymoses, cyanosis and petechiae.

Another case was investigated by Dr OG Williams (Swansea, Wales). A 48-year-old man was found in a derelict house, hanging from beams in the broken ceiling. He had, presumably deliberately, hooked his knees over one beam and secured his feet under the adjacent beam, so that his body was hanging vertically downwards, as a means of suicide. Although some postmortem decomposition was present, autopsy revealed no other cause of death, other than the inverted posture. There was a large collection of blood-stained fluid beneath the scalp, due to gravitational movement of blood and tissue fluid.

In a further case, a 46-year-old man was found in a head-down position at the bottom of a narrow staircase after obviously falling down the stairs (Figure 24.2). His head was wedged between the last step and a closed sliding door. Autopsy revealed craniofacial blunt force injuries, a non-dislocated fracture of the fourth cervical vertebral body (with intact ligaments of the spine and cervical cord) and massive cerebral and pulmonary oedema. Although the heart was significantly enlarged (610 g), the coronary arteries showed only minor arteriosclerotic changes. The alcohol concentration measured was 2.06 parts per thousand in blood and 2.6 parts per thousand in urine. The alcohol intoxication increased the risk of falling and, together with the trauma of the cervical spine, made it impossible for the man to free himself, so he ultimately died in a head-down position.

As vital signs, disseminated centrilobular necrosis and steatosis in the liver tissue and cardiac contraction bands were observed (Figures 24.3 and 24.4) [6,7,13,22,28,32–35,36,44,48–52,56,61].

Several further cases have been reported and are summarized in Tables 24.2 and 24.3 (modified according to Doberentz and Madea [20] and Sauvageau et al. [48]).

## Discussion

The following mechanisms of death must be discussed:

1. Asphyxia due to fixation of the chest with hindering of thoracic excursions and ventilation.
2. Circulation failure due to the head-down position. This second factor may be the prevalent one in cases of hanging by the feet in a head-down position – see the cases reported by Purdue [47] and Marshall [36].

The fibres of the sack in Case 2 were already extremely stretched. Thoracic excursions were probably only possible between end expiratory position and expiratory resting position. Compensation of the extremely hindered costal ventilation by abdominal ventilation (diaphragmatic ventilation) was probably only very limited, since the abdominal wall was fixed within the sack.

By the fixation of the thorax, the elastic resistance of the pulmonary system was raised considerably. Consequently, the compliance decreased. The respiratory effort against the elastic resistance increased considerably, over-proportionally to the respiratory effort provided by the energy demand of respiratory muscles [43].

This asphyxia due to increased respiratory resistance is worsened by the extreme oxygen demand of the respiratory muscles. In cases of hard physical work, 20 per cent of the oxygen uptake is used for respiratory work, compared to only 2 per cent in normal respiration [55].

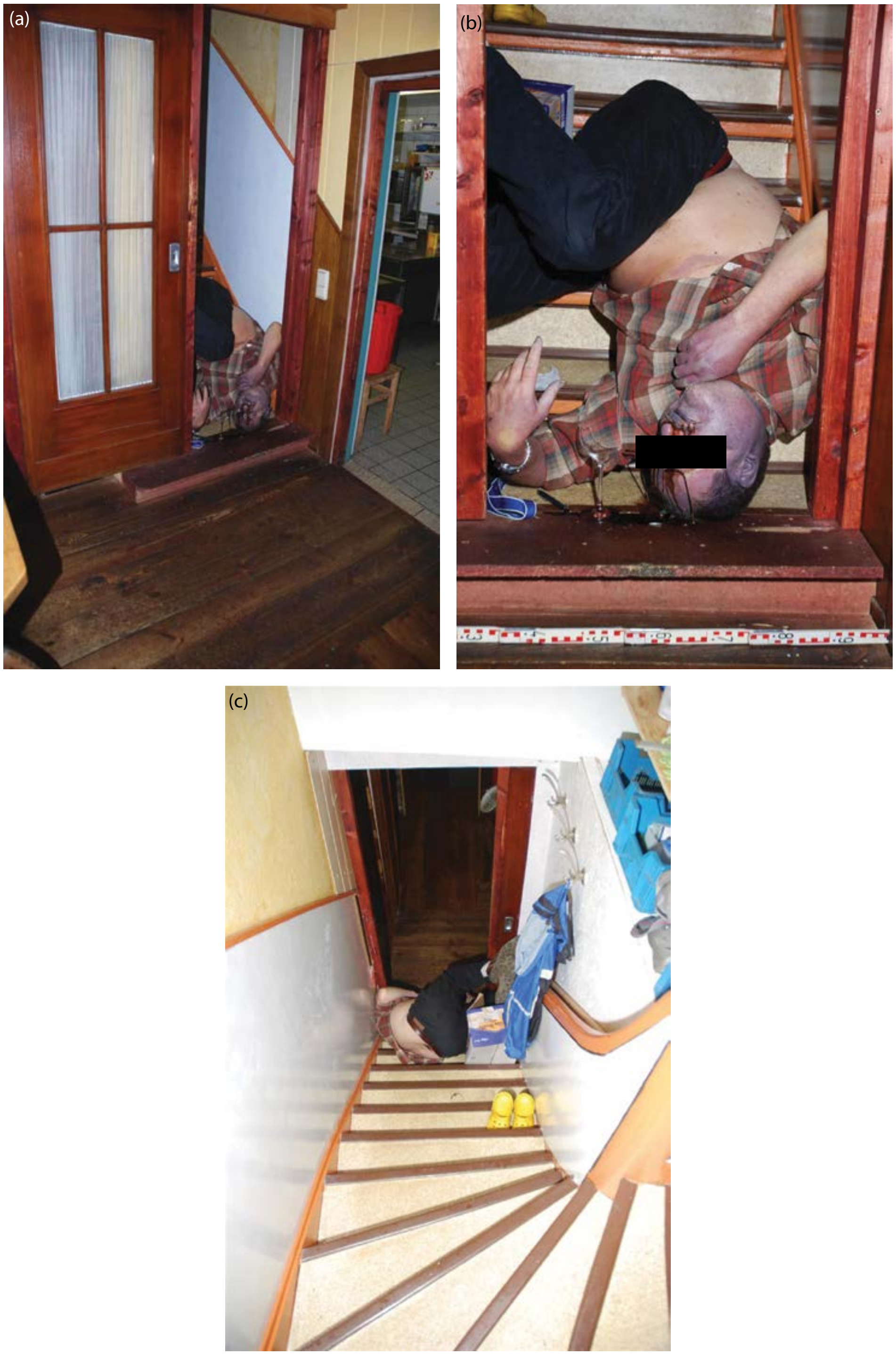
Circulatory failure as a cause of death in a head-down position may be due to several factors. Investigations on young healthy men revealed that, in orthograde (feet down) suspension (body fixed at the arms), orthostatic collapse follows after only 12 minutes; after 6 minutes there is a drop of blood pressure from 120 mmHg to 70 mmHg and the ECG indicates lack of extra coronary oxygen [9,40,60].

Reports on the circulatory regulation in a head-down position are almost absent from the literature. Physiology and cardiology have been studied mainly in the circulatory regulation of supine persons, standing persons and the postural change from supine to standing, but in aerospace medicine, physiological investigations on circulatory regulation in a head-down position have been carried out [23,24,60].

According to a paper by Althoff et al. [1], they were warned by clinicians and physiologists not to perform investigations on circulatory regulation in a head-down position because, in a long-lasting head-down position, the hydrostatic pressure in the venous system could increase to 100 mmHg and the danger of micro- or macrohaemorrhages in the central nervous system could not be excluded. Experimental investigations on afebrile, normotensive hospital patients, convalescent from minor ailments, were, however, carried out by Wilkins et al. [60] on 42 persons for 2–30 minutes without development of cerebral haemorrhages.

Our knowledge of circulatory regulation in supine and standing persons allows us to formulate several hypotheses.

The hydrostatic indifference level (i.e. the horizontal level within the human body where, in postural changes from supine to standing, the pressure within the vessels (and the diameter of the vessels) does not change) in humans is at the height of the liver, 10 cm below the diaphragm [12].



[redacted]

**Figure 24.2** (a) The finding position at the bottom of a steep staircase. The door was originally closed. (b) The finding position at the bottom of the stairs. Note the congestion of the face and upper trunk. There is a broken glass beside the head. (c) The finding position at the bottom of steep stairs.

Above this hydrostatic indifference level, the hydrostatic pressure decreases; below it, it increases (Figure 24.5).

In a head-down position it is completely reversed: above the hydrostatic indifference level (i.e. in the thorax, neck and head), the hydrostatic pressure increases; below it (in the legs), it decreases. However, it remains unclear whether the hydrostatic indifference level in a head-down position is the same as in postural changes from a supine to upright position.

As a consequence of the increased hydrostatic pressure in the thorax, head and neck, the pressure in the venous system rises (e.g. in the internal jugular vein [60]). This is

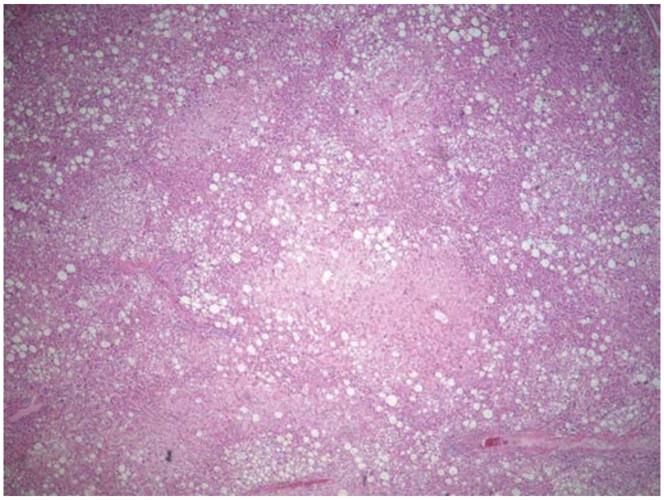


**Figure 24.3** Liver tissue with disseminated centrilobular necrosis and steatosis (H&E stain ×4).

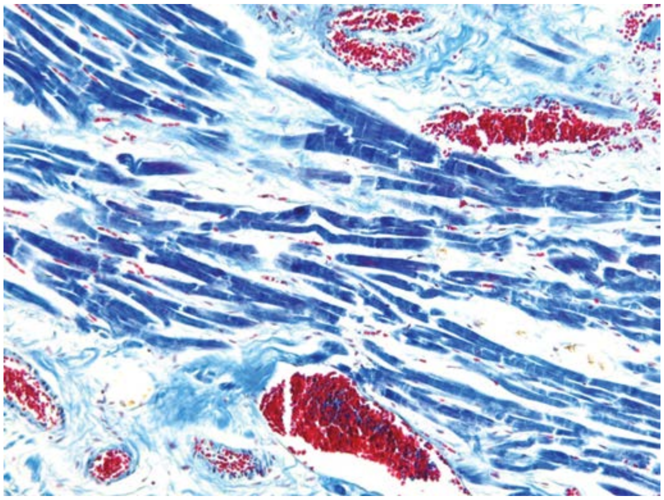


**Figure 24.4** Cardiac tissue with disseminated contraction bands (CAB stain ×20).

followed by a blood-volume shift into regions of the body where mechanisms which promote the venous return of blood to the heart are insufficiently developed.

It is not known whether (and in which direction) the raised transmural pressure in the venous part of cerebral circulation (mainly the thrombencephalon circulatory centres) affects the nervous regulation of circulation.

By the addition of the hydrostatic pressure to the arterial blood pressure in cranial arteries in a head-down position, theoretically (via the carotid system) the heart frequency and arterial blood pressure may be decreased. Experimental investigations by Wilkins et al. [60] revealed a rise of blood pressure in the cranial arteries due to hydrostatic effects which were followed by subsequent normalization (Table 24.4).

Results compiled from the literature on human experimental studies [10,11,14,15,18,25,26,29–31,38,41,42, 48,58,60] with head-down tilt of more than 30 degrees was published by Sauvageau et al. [48] and is shown in Table 24.5.

**Table 24.2 Case reports from the literature of deaths in a head-down position (according to Sauvageau et al., 2008)**

| Author(s) [Reference] | Number of cases | Gender | Age (years) | Circumstances | Alcohol intoxication |
|---|---|---|---|---|---|
| Schäfer [50] | 1 | ♂ | Middle-aged | Body lying lengthwise in the staircase, head in the lowest position | + |
| | 1 | ♂ | Elderly | Head-down in a narrow hole on the ground of a building site | + |
| | 1 | ♂ | 77 | Supine position on a table, head and lower legs hanging down | – |
| Madea [33] | 1 | ♂ | 56 | Head-down position, hanging in a sack | – |
| | 1 | ♀ | 85 | Left heart failure on severe aortic stenosis after a 30-degree tilt for barium enema | ? |
| | 2 | ♂ | nd | 'Jack-knifed' across the hips, head and upper body hanging down | nd |
| | 2 | nd | nd | Partially fallen from bed with head on floor and legs still in the bed | + |
| | 1 | ♂ | 48 | Hanging from beams in a broken ceiling, head down | nd |
| Purdue [47] | 1 | ♂ | 48 | Right foot stuck between two spikes of a fence | + |
| Falk et al. [22] | 1 | ♀ | 29 | On a bar stool, body retroflected in lumbar region, head hanging down | + |
| Marshall [36] | 1 | ♂ | 67 | Suspended by his clothing upside down in a thorn hedge | + |
| Yoshida et al. [61] | 1 | ♂ | 74 | Head-down position in a hole in a pile of rubbish | – |
| Lawler [32] | 1 | ♂ | 62 | Right foot trapped beneath part of broken boarding | – |
| Thiel and Huckenbeck [56] | 1 | ♂ | 64 | Hanging head down from a railing, right knee jammed between wall and railing | nd |
| Sauvageau et al. [48] | 1 | ♀ | 84 | Body free suspended downwards with the right leg stuck in the railings of the staircase | – |
| Doberentz and Madea [20] | 1 | ♂ | 67 | Found in a head-down position at the bottom of a steep staircase | + |

*Abbreviation:* nd, No data.

**Table 24.3** Case reports from the literature of deaths in a head-down position (according to Doberentz et al., 2009)

| Author(s) [Reference] | Victim age/gender | Incident | BAC (‰) |
|---|---|---|---|
| Marshall [36] | 11 months | Hanging in a head-down position in a bed | |
| | 67 years/male | Suspended by his clothing upside down in a thorn hedge | 2.3 |
| | | Hanging on the feet in World War II | |
| Hilgermann and Richter [27] | 72 years/female | Autoerotic accident | |
| Prokop and Radam [46] | Old woman | Hanging out of a window in a head-down position | |
| Madea et al. [35] | 56 years/male | Autoerotic accident, hanging in a sack | |
| Purdue [47] | 48 years/male | Hanging on a fence | 1.29 |
| Lawler [32] | 62 years/male | Body suspended with right foot trapped beneath part of a broken board | |
| Madea and Williams [pers. comm.] | 48 years/male | Hanging upside down with the backs of the knees across a beam | |
| Madea and Knight [pers. comm.] | 20–30 years/male | Upper part of the body hanging upside down through a fanlight | |
| | 40 years/male | Hanging in a fanlight | 1.1 |
| Yoshida et al. [61] | 74 years/male | Hanging in a hole | |
| Iannaccone et al. [28] | 35 years/male | Hanging in a fork of a tree | |
| Thiel and Huckenbeck [56] | 64 years/male | Hanging over a railing | |
| Schäfer [50] | 77 years/male | Head-down position on a table | |
| | Middle-aged male | Head-down position on a stair | 2.80 |
| | Older male | Hanging in a hole | 2.76 |
| De Donno et al. [17] | 78 years/male | Hanging in a bush | 2.1 |
| Sauvageau et al. [48] | 82 years/female | Freely suspended downward with the right leg stuck in the railings of a staircase | |
| Doberentz and Madea [20] | 46 years/male | Found in a head-down position at the bottom of a staircase | |

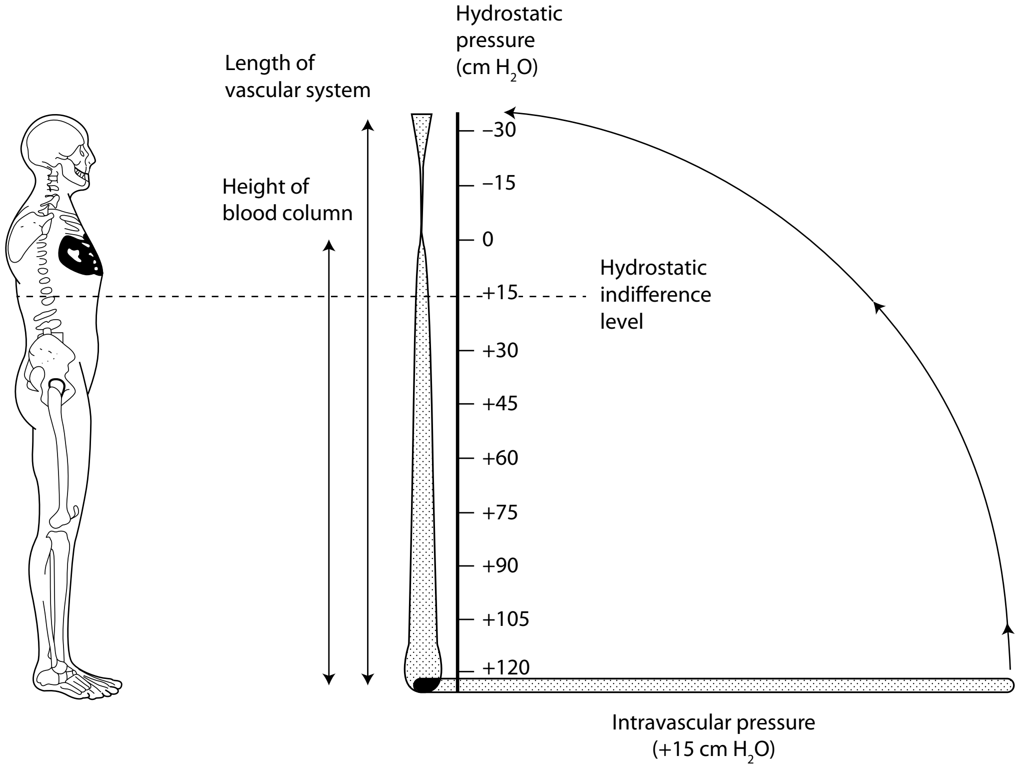

Length of vascular system
Hydrostatic pressure (cm $H_2O$)
Height of blood column
−30
−15
0
+15
Hydrostatic indifference level
+30
+45
+60
+75
+90
+105
+120
Intravascular pressure (+15 cm $H_2O$)

**Figure 24.5** Hydrostatic indifference level within the human body. In postural changes from horizontal to upright, the pressure and the diameter of venous vessels in the hydrostatic indifference level remain unchanged (according to Busse) [12].

**Table 24.4 Subjective symptoms and effects of head-down position (according to experimental observations from Wilkins et al., 1950)**

| Effects of head-down position | Observations |
|---|---|
| Subjective symptoms | Sensation of tumbling head-over-heels<br>Tensing of the body musculature<br>Facial congestion/cranial swelling<br>Congestion of nasal mucosa<br>Perspiration of neck and face<br>Tears from eyes |
| Effects on arterial blood pressure | Fall of blood pressure in femoral artery<br>Rise (especially of diastolic pressure) in brachial artery (or no change); first shift due to passive hydrostatic effects, then decline in pressure of both arteries<br>Irregularity of pulse beats and decrease of pulse rate from 90–100 to 44–68 beats/min |
| Effects on internal jugular venous pressure | Immediate rise of venous pressure (hydrostatic effect) followed by a continuous rise of the same amount (drainage of blood from the elevated parts into the dependent chambers) |
| Effects on cardiac output | Increased cardiac output |

**Table 24.5 Results complied from the literature on human experimental studies with head-down tilt (HDT) of more than 30 degrees****

| IN HEALTHY YOUNG INDIVIDUALS | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Ref.** | **HDT*** | **Time** | **# cases** | **Ratio M:F** | **Age** | **SV** | **HR** | **CO** | **PR** | **CR** | **VP** | **MP** | **SP** | **DP** | **$SaO_2$** | **$pCO_2$** |
| *From supine to HDT* | | | | | | | | | | | | | | | | |
| [10] | 30° | 5 min | 12 | 12 M | 24.6 ± 1.7 | | = | | | | | = | = | = | | |
| [11] | | | 12 | 12 M | 22.5 ± 0.39 | ↑ | ↓ | ↑ | ↓ | | | ↓ | = | ↓ | | |
| [12] | | | 20 | 20 M | 18–20 | | = | | | | | = | = | = | | |
| [13] | | 10 min | 12 | 12 M | 25.2 ± 2.2 | ↑ | = | ↑ | ↓ | | ↑ | = | = | = | | |
| [14] | | | 19 | 8 M: 11 F | nd | | = | | ↓ | ↑ | | ↓ | | | | |
| [15] | | | 32 | 18 M: 14 F | 20 ± 0.84 | | ↑ | | | | | | = | = | = | |
| [10] | 60° | 5 min | 12 | 12 M | 24.6 ± 1.7 | | ↓ | | | | | = | ↓ | = | | |
| [12] | | | 20 | 20 M | 18–20 | | = | | | | | ↑ | = | ↑ | | |
| [16] | 70° | 5 min | 12 | 12 M | 19–24 | | ↑ | ↑ | | | | ↑ | ↑ | ↑ | | |
| [17] | 75° | 2 to 30 min | 42 | 40 M: 2 F | nd | ↑ | initial ↓, then = | ↑ | | | ↑ | ↑ | ↓ | ↓ | | |
| [18] | 80° | 1 min | 13 | 11 M: 2 F | 26.7 ± 5.4 | | initial ↓, then = | | | | | = | | | | = |
| [12] | | 5 min | 20 | 20 M | 18–20 | | = | | | | | ↑ | = | ↑ | | |
| [10] | 90° | 5 min | 12 | 12 M | 24.6 ± 1.7 | | = | | | | | = | = | = | | |
| *From sitting or upright to HDT* | | | | | | | | | | | | | | | | |
| [19] | 30° | 5–7 min | 10 | 8 M: 2 F | 19–22 | | ↓ | | ↓ | | | ↑ | | | | |
| [20] | 45° | 2 min | 9 | 6 M: 3 F | 40 ± 13 | | ↓ | | | | | | | | = | |
| [15] | 75° | 10 min | 32 | 18 M:14 F | 20 ± 0.84 | | ↓ | | | | | | = | = | | |
| [21] | | 10 min | 21 | nd | 24.6 ± 3 | | ↓ | | | | | ↓ | = | ↓ | | |
| [17] | | 2 to 30 min | 42 | 40 M: 2 F | nd | ↑ | ↓ | ↑ | ↓ | | ↑ | ↓ | ↓ | ↓ | | |
| [22] | 90° | 1 à 10 min | 8 | 8 M | 23.4 ± 0.4 | ↑ | = | ↑ | | | | = | = | = | | |
| **IN OLDER INDIVIDUALS** | | | | | | | | | | | | | | | | |
| **Ref.** | **HDT*** | **Time** | **# cases** | **Ratio M:F** | **Age** | **SV** | **HR** | **CO** | **PR** | **CR** | **VP** | **MP** | **SP** | **DP** | **$SaO_2$** | **$pCO_2$** |
| *From supine to HDT* | | | | | | | | | | | | | | | | |
| [10] | 30° | 5 min | 12 | 12 M | 68.6 ± 2.2 | | = | | | | | = | = | = | | |
| | 60° | 5 min | 12 | 12 M | 68.6 ± 2.2 | | = | | | | | ↑ | ↑ | = | | |
| | 90° | 5 min | 12 | 12 M | 68.6 ± 2.2 | | ↑ | | | | | ↑ | ↑ | ↑ | | |
| *From sitting or upright to HDT* | | | | | | | | | | | | | | | | |
| [21] | 30° | 10 min | 19 | nd | 65.7 ± 6 | | ↓ | | | | | ↓ | = | ↓ | | |

*Abbreviations:* M, male; F, female; SV, stroke volume; HR, heart rale; CO, cardiac output; PR, peripheral resistance; CR, cerebral resistance; VP, central or jugular venous pressure; MP, mean arterial blood pressure; SP, systolic blood pressure; DP, diastolic blood pressure; $SaO_2$ oxygen saturation; nd, no data.

* Head-down tilt from the horizontal plane (0°).

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**Table 24.6 Haemodynamic dysregulation in a head-down position**

| Hydrostatic dysregulation | Effect |
|---|---|
| Hydrostatic pressure in 'upper' body part | 'Shift' of blood into intrathoracic blood pools |
| Transmural pressure in the venous part of cerebral circulation | |
| Raised static pressure in carotid sinus | Drop of arterial blood pressure |
| Decreased venous return of blood to the heart | Fall away of muscular venous pump<br>Fall away of the suction pressure effect of respiration |

The hindering of ventilation itself also has consequences for the regulation of circulation. With deep inspirations being impossible, the suction-pressure effect of ventilation decreases and the venous return of blood to the heart is hindered.

Thus, in a head-down position, the following haemodynamic effects are to be expected (Table 24.6):

- There is marked depletion of the venous return of blood to the heart.
- There is an increase of transmural pressure in the venous part of cerebral circulation
- There is a probable decrease of systemic (arterial) blood pressure (but here some complex regulatory mechanisms are possible). In many cases of positional asphyxia and also death in a head-down position alcoholization plays an important role. Alcoholization increases the risk of haemodynamic dysregulation [2,3,31,53,54,57].

How long does it take to die in a head-down position? Older reports speak of several hours or even a day. The marked infiltration of polymorphonuclear phagocytes in the cuff-like haemorrhages of the forearm in Case 2 indicate a terminal episode of some hours.

## Conclusion

In deaths in uncommon body postures, autopsy often reveals no clear anatomical cause of death, although it is evident that the deceased has come into the position alive and the position contributed or led to death. Case histories on deaths in a head-down position are rare, but the problem may be of increasing interest in connection with torture, and the forensic pathologist may be asked how long it might take for a person to die in this position. From the limited experience of some recent case histories and the experimental investigations of Wilkins et al. [60], it can be said that, in healthy individuals, it may take some hours.

A number of common features of deaths in a head-down position have been noted by Sauvageau et al. [48]:

1. Finding of the body in a head-down or inverted position.
2. Marked 'monstrous' congestion of the head, neck and other dependent parts of the body.
3. Congestion and oedema of the brain and lungs.
4. Lack of a definite pathoanatomical cause of death.
5. Death by a head-down position is an exclusion diagnosis that can only be put forward after an extensive case work-up, because it is not merely because a person is found head-down that the position itself is the cause of death.
6. Death by head-down position should not be confused with the more common positional asphyxia.
7. Human experimental studies suggest that the pathophysiology of true deaths by head-down position is more related to heart failure than to asphyxia.

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# 25 Traumatic Carotid Sinus Reflex

*Elke Doberentz and Burkhard Madea*

## Introduction

The carotid body and carotid sinus are localized in the area of the carotid bifurcation and respond to pressure fluctuations in the arterial blood vessel system. In case of irritation or stimulation, nervous impulses can reflexively increase the ventilation or slow down the heart rate and blood pressure, respectively. The external stimulation of the carotid sinus by neck compression with subsequent bradycardia or asystolia, especially in pre-existing heart disease, is discussed controversially in the literature. Histological examination of the tissue of the carotid bifurcation, particularly with regard to haemorrhage as an indication of tissue trauma, is simple and feasible and should be carried out in routine diagnostics.

In 20 (43) cases of violence against the neck and an additional 82 cases in a control group without neck trauma and variable causes of death, the carotid bifurcations were examined histologically [10]. Haemorrhage was found in only one case of violence against the neck, which suggested a direct trauma to the tissue of the carotid bifurcation, but evidence of lethal cardiac reflex was not found in any case.

The centre of circulation and respiration is localized in the medulla oblongata. The cardiocirculatory activity is mainly regulated by the autonomic nervous system. The carotid body and carotid sinus are involved in this regulation (Figure 25.1). The carotid body is a rice-grain-sized knot (Figure 25.2). It is localized outside the arterial wall in the carotid bifurcation between the external and internal carotid artery [1,6,16]. It is directly connected with the vascular lumen (Figure 25.3). The location and also the shape of the carotid bodies can vary [34]. The carotid body induces a reflexively vagal mediated hyperventilation with increased ventilator frequency and volume in cases of reduction of arterial oxygen tension ($pO_2$), of systemic hypoxia and rise of arterial carbon-dioxide tension ($pCO_2$) as well as a decrease of pH values. It is also stimulated by falling arterial pressure. The response is a peripheral constriction of the blood vessels by stimulating sympathetic fibres [7,13]. In this way the carotid body helps to regulate the blood pressure [7].

Of high forensic interest is the carotid sinus, which is located in the medial vessel wall of the internal carotid artery in the area of the carotid bifurcation. The carotid sinus consists of baroreceptors (stretch-sensitive fibres), which are located in the adventitia as small bundles of nerve fibres (Figure 25.4). They are in contact with elastic fibres of the vessel wall [4,22]. The carotid sinus is stimulated by elongation and deformation of the carotid wall, especially in response to rapidly increasing blood pressure (rise of intravascular pressure) and also external pressure. By rapid and powerful reflex stimulation of the parasympathetic area of the medulla oblongata and an inhibition of sympathetic areas, this induces a decrease in heart rate (bradycardia) and in blood pressure (hypotonia) and the reduction of the peripheral vessel tone (vasodilatation) [7]. Alterations in an electrocardiogram occur after a few seconds after stimulus onset [7,3,8]. In contrast, the compression of the common carotid artery beneath the carotid bifurcation is accompanied by an acute reduction of vascular pressure due to decreased blood flow. This induces tachycardia, tachypnoea and the release of catecholamines [18]. The faster the pressure changes, the more intense the reactions are.

Afferent fibres of the carotid body and the carotid sinus join the thyroglossopharyngeal nerves and project to the medulla oblongata. Efferent fibres project to the vagi nerves [36]. The carotid body and carotid sinus are interacting [7].

There is no question that stimulation, especially of the carotid sinus, can induce bradycardia up to asystolia with circulatory failure. From a forensic point of view, it is of particular importance if a (minor) trauma of the neck can cause sudden loss of consciousness and death due to carotid sinus reflex, for example in a case of violence against the neck without the aim of killing the victim. If a lethal cardiac reflex is assumed, it has to be proved. In cases of death due to violence against the neck, therefore, histological findings should be analyzed as a possible direct sign of tissue trauma in consideration of macroscopical findings (especially with regard to haemorrhage as a sign of tissue trauma) and history, which would point to a cardiac reflex and help prove its occurrence.

## Material and methods

The study group consisted of 20 cases with verified violence against the neck, 8 female and 12 male victims. The mean age was 41.8 years (18–81 years). The group consisted of 14 cases of death by hanging, 2 cases of violence against the neck (blows and kicks), 3 cases of ligature strangulation and 1 case of manual strangulation (Table 25.1). All available information was collected

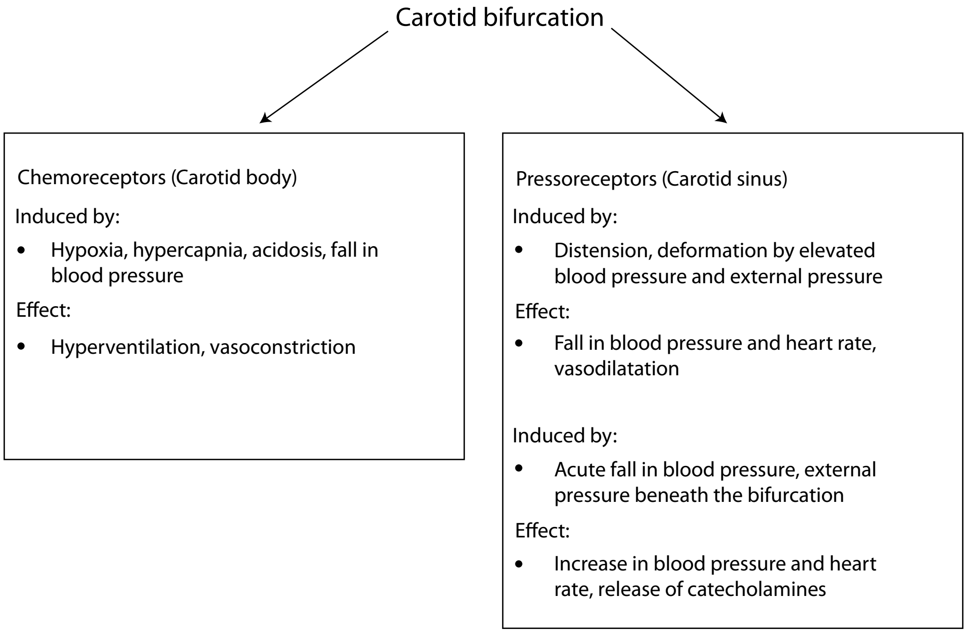
Carotid bifurcation
Chemoreceptors (Carotid body)
Induced by:
• Hypoxia, hypercapnia, acidosis, fall in blood pressure
Effect:
• Hyperventilation, vasoconstriction
Pressoreceptors (Carotid sinus)
Induced by:
• Distension, deformation by elevated blood pressure and external pressure
Effect:
• Fall in blood pressure and heart rate, vasodilatation
Induced by:
• Acute fall in blood pressure, external pressure beneath the bifurcation
Effect:
• Increase in blood pressure and heart rate, release of catecholamines

**Figure 25.1** Diagram of the regulation of respiration and circulation by the carotid body and sinus.

from autopsy protocols or from the documentation from investigation authorities in terms of rope width, location of the knot, drop height, etc.

The control group consisted of 82 cases with natural and non-natural manners of death (e.g. myocardial infarction or drug intoxication) with different agonal periods but without evidence of trauma against the neck. In the group, 55 victims were female and 27 male. The mean age was 55.2 years (17–85 years).

The left and right carotid bifurcations were collected at routine autopsies. The neck organs were dissected in a bloodless situation *in situ*. In each case the two common carotids, including the carotid bifurcations, were excised. The common carotid was cut 2 cm below the bifurcation and the internal and external carotid arteries were transected 2 cm above the carotid bifurcation. The excised carotid bifurcations surrounded by tissue were fixed in formalin. After fixation, the carotid bifurcations were laminated in thin slices of a few millimetres in the horizontal plane (Figure 25.5). The tissue was embedded in paraffin wax. Haematoxylin-eosin (H&E) and Azan staining

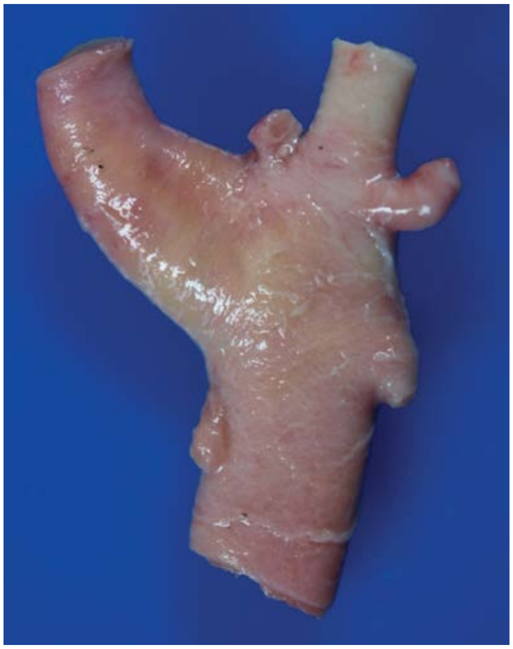

**Figure 25.2** The carotid bifurcation (for the image skeletonized) with the clearly visible carotid body in the distribution of the internal (left side) and external carotid artery (right side).

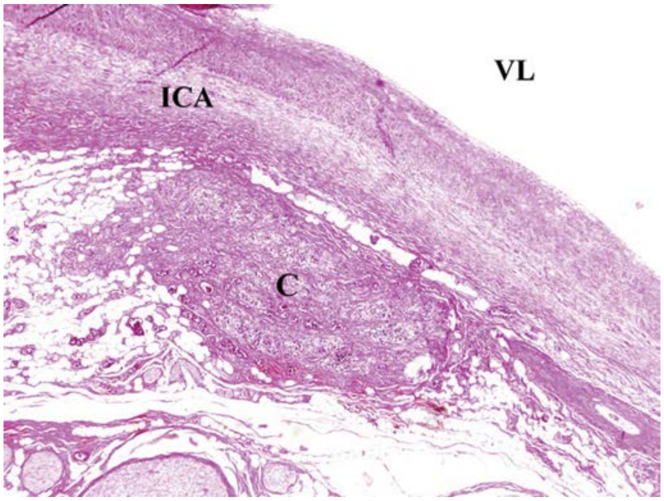

**Figure 25.3** Carotid body (C) outside the internal carotid artery (ICA); VL, vascular lumen (H&E, ×10).

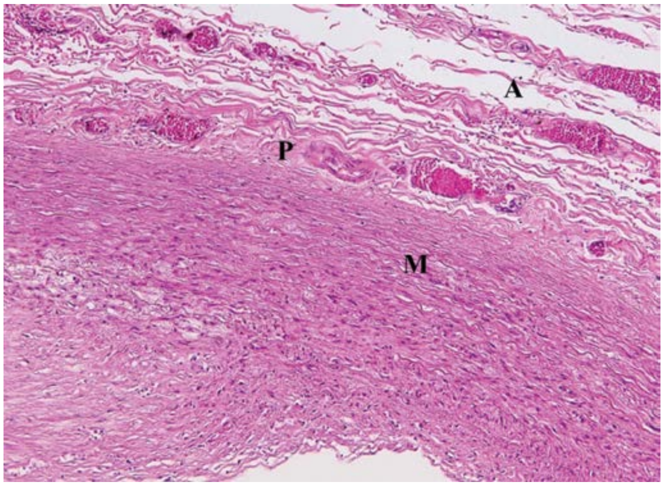

**Figure 25.4** Carotid sinus. Nerve fibres of pressoreceptors (P) between adventitia (A) and media (M) of the vessel wall of the internal carotid artery (H&E, ×40).

were performed. All carotid bifurcations were examined microscopically using a light microscope.

In the study group, histological examinations of the lungs and brain were also performed, as well as toxicological analysis.

## Results

In the control group, haemorrhage in the tissue of the carotid bifurcation could not be detected. A wide range of different states of arteriosclerosis of the vessel walls was found in this group (ages between 17 and 85 years) due to pre-existing illness and varying ages.

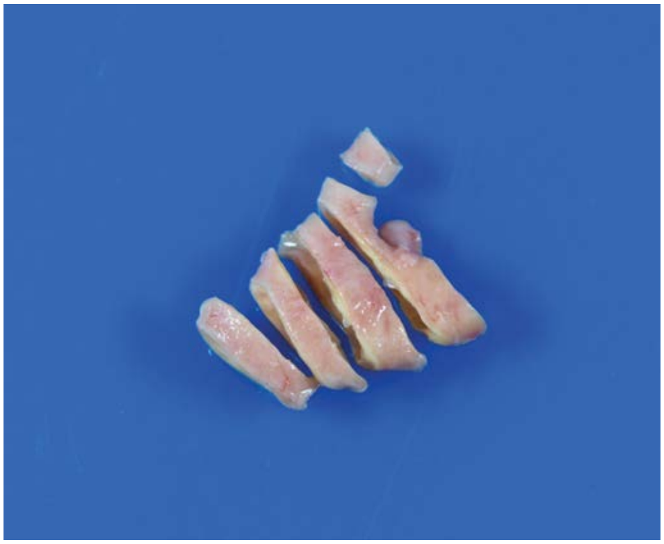

**Figure 25.5** Material and methods: Sampling of carotid bifurcation. The carotid bifurcation is cut into lamellar pieces, which are stained (H&E, Azan, ferric) and examined by light microscopy.

In all of the study group cases, violence against the neck had occurred in different degrees of severity and forms and this was reflected in the different findings (Table 25.1). In all 14 cases of hanging, haemorrhage was found in the tissue of the neck. Two cases of violence against the neck, three cases of ligature strangulation and one case of manual strangulation showed haemorrhage in the tissue. Fractures of the larynx and hyoid bone were found in two cases of hanging, in none of the cases of violence against the neck, in two cases of ligature strangulation, and in the case of manual strangulation.

In six of the cases of hanging, signs of venous stasis (petechiae) were present (blue hanging) and all cases showed histological vital reactions. All the cases of ligature strangulation showed petechiae, as did the case of manual strangulation.

Histological examination revealed haemorrhagic pulmonary oedema and/or cerebral oedema in all cases. In only one case of the study group (case 20) was haemorrhage found in the tissue of the carotid bifurcation.

In the study group severe arteriosclerosis with plaques of the vessels was not found. In two cases (cases 2 and 7) the heart weight exceeded the critical weight of 500 g. In case 1, relevant stenosis of a coronary vessel was found. Furthermore, the heart muscle did not show pathological alterations in any case.

In three cases (cases 1, 4 and 5) signs of venous stasis or prolonged hypoxia were not found, but there were signs of preserved blood circulation in the form of haemorrhage in the musculature or oedema as a vital sign.

These results were confirmed in a larger study group ($n = 43$, age 11–87 years, 14 female, 29 male) with known trauma to the neck: 35 hanging, 5 ligature strangulation, 2 hitting and kicking, 1 throttling.

Haemorrhages into the tissue around the carotid bifurcation were found in only one case of ligature strangulation (see Figures 25.7–25.9).

## Discussion

Violent actions against the neck may be hanging, manual or ligature strangulation, strangling, beating, kicking and compression of different types and they can produce variable injuries on the neck [25]. Peripheral arterial chemoreceptors and pressoreceptors, which are localized in the carotid bifurcation, are involved in the regulation of respiration and circulation. In many publications the relevance of death due to carotid sinus reflex in case of violence against the neck is discussed controversially [29,31].

Due to the anatomical location of the carotid arteries (Figure 25.6), they can be pressed against the spine in case of lateral blunt force trauma. Furthermore, the larynx can spread apart in case of frontal trauma and can affect the carotid arteries [1]. When hanging, the typical location of the ligature mark above

**Table 25.1 Detailed list of the study group cases with verified lethal violence against the neck**

| No. | Age | m/f | Cause of death | Findings of the neck | Signs of venous stasis of the head | Heart | Macro- and histomorphological findings brain/lungs | BAC ‰ |
|---|---|---|---|---|---|---|---|---|
| 1 | 52 | f | Profound sharp force injury, blunt force injury | Haemorrhage around the larynx | None | 320 g, stenosis of the RIVA | – | 0.03 |
| 2 | 48 | f | Typical hanging with a belt, found in a squatting position | Ligature mark of the skin | Petechiae | 520 g | Interstitial-alveolar oedema, cerebral oedema | 0.00 |
| 3 | 53 | m | Ligature strangulation with a computer cable | Haemorrhage into the right sternocleidomastoid muscle | Petechiae | 450 g, moderate arteriosclerosis | – | 2.25 |
| 4 | 51 | m | Hanging with a rope | Ligature mark above the larynx | None | 470 g | Mild pulmonary oedema, cerebral oedema | 0.01 |
| 5 | 18 | f | Violence against the neck (blows and kicks) | Severe haemorrhage in the muscles and all layers of the skin | None | 25 g | – | 0.02 |
| 6 | 53 | m | Hanging with a dog lead | Ligature mark of the skin | None | 730 g | Interstitial-alveolar oedema, cerebral oedema | 1.53 |
| 7 | 49 | m | Atypical hanging with a 3 cm wide belt | None | Petechiae | 550 g, moderate arteriosclerosis | Interstitial-alveolar oedema, cerebral oedema | 0.05 |
| 8 | 39 | m | Hanging with a rope | Ligature mark of the skin | Petechiae | 350 g | No pulmonary oedema, cerebral oedema | 1.15 |
| 9 | 26 | m | Hanging with a rope | Ligature mark of the skin | None | 380 g | Interstitial oedema, no cerebral oedema | 0.00 |
| 10 | 27 | m | Ligature strangulation | Ligature mark of the skin, mild haemorrhage of both sternocleidomastoid muscles, fracture of the right superior horn of the thyroid cartilage | Petechiae | 350 g | Interstitial oedema, pulmonary oedema | 0.00 |
| 11 | 31 | m | Hanging with a belt fixed at 42 cm in height | Ligature mark of the skin | None | 380 g | Alveolar oedema, mild cerebral oedema | 0.01 |
| 12 | 45 | m | Hanging with a belt at the door frame, belt buckle on the left side of the neck | Fracture of the both superior horns of the thyroid cartilage, fracture of the right horn of the thyroid bone without haemorrhage | Petechiae | 280 g | Interstitial-alveolar oedema, cerebral oedema | 0.00 |
| 13 | 81 | f | Manual strangulation | Bruising of the skin on the right cervical side, fracture of the both superior horns of the thyroid cartilage, fracture of both horns of the hyoid bone with massive haemorrhage | Petechiae | 310 g – moderate arteriosclerosis | No pulmonary oedema, cerebral oedema | 0.00 |

*(Continued)*

**Table 25.1 (*Continued*)** **Detailed list of the study group cases with verified lethal violence against the neck**

| No. | Age | m/f | Cause of death | Findings of the neck | Signs of venous stasis of the head | Heart | Macro- and histomorphological findings brain/lungs | BAC ‰ |
|---|---|---|---|---|---|---|---|---|
| 14 | 36 | m | Hanging with a belt at the door frame | Ligature mark of the skin | None | 440 g | Interstitial-alveolar oedema, cerebral oedema | 0.02 |
| 15 | 53 | f | Hanging with a rope found in a seated position, knot on the left side of the neck | Ligature mark of the skin, fracture of both the superior horns of the thyroid cartilage, fracture of the right horn of the hyoid bone without haemorrhage | Petechiae | 550 g | Interstitial oedema, cerebral oedema | 1.67 |
| 16 | 32 | m | Typical hanging with a rope, feet on the ground | Ligature mark of the skin | None | 360 g | Interstitial oedema, cerebral oedema | 1.43 |
| 17 | 42 | f | Hypoxic brain damage due to hanging | None | None | 440 g | Alveolar oedema, cerebral oedema | 0.00 |
| 18 | 38 | f | Hypoxic brain damage due to hanging | Haemorrhage into the platysma | None | 275 g | No pulmonary oedema, cerebral oedema | 0.02 |
| 19 | 27 | m | Hanging with a belt at the door frame, belt buckle on the left side of the neck | None | Petechiae | 300 g | Interstitial oedema, cerebral oedema | 1.6 |
| 20 | 35 | f | Ligature strangulation | Ligature mark supralaryngeal, haemorrhage into the subcutaneous tissue and anterior musculature of the neck, haemorrhage on the right and back of the larynx, fracture of the right horn of the hyoid bone, fracture of the superior horn of the thyroid cartilage, petechiae of the pharynx | Engorged cyanotic face, petechiae | 300 g | Mild cerebral oedema, no pulmonary oedema | 0.16 |

*Abbreviations:* BAC, Blood alcohol concentration; m, male; f, female.

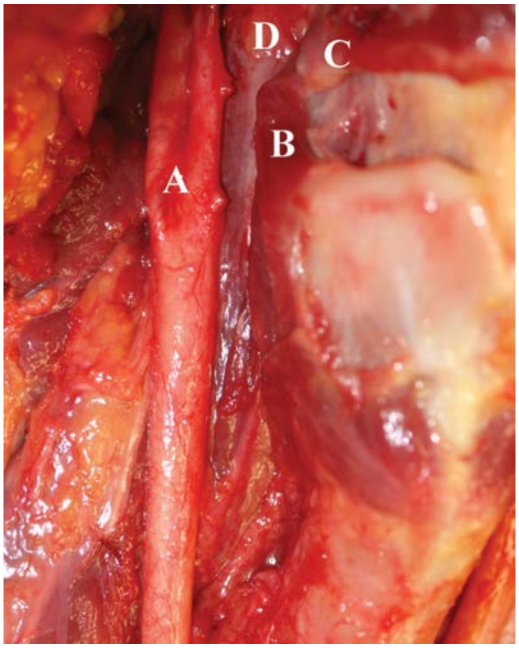

**Figure 25.6** Anatomical location of the right common carotid artery with the carotid bifurcation (A), in direct association with the right superior horn of the thyroid cartilage (B), the hyoid bone (C) and the cervical spine (D).

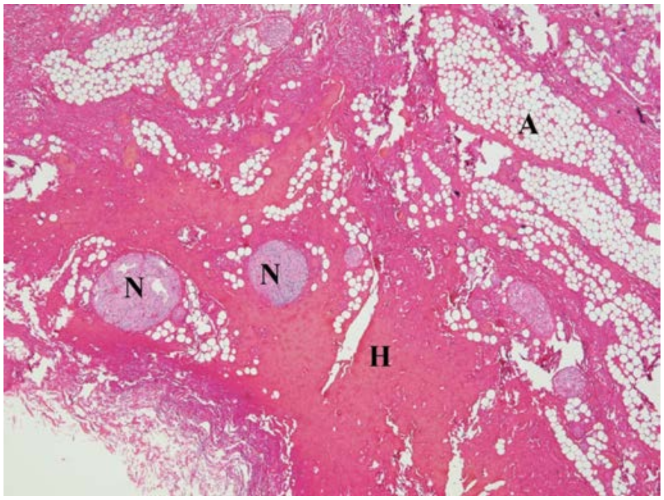

**Figure 25.7** Case of ligature strangulation in the study group (case 20): haemorrhage (H) in the connective and adipose tissue (A) of the carotid bifurcation, left carotid bifurcation; N = nerves (H&E, ×10).

the larynx can cause pressure on the carotid bifurcation with initial bradycardia and asystolia [26], but ventricular rhythm can return. In cases of ligature strangulation, the throttle mark is often localized below the carotid bifurcations. Direct pressure on the carotid sinus does not exist, but the reduction of blood flow above the strangulation area, a vagal depression and sympathetic activation can result [18]. In cases of manual strangulation, variable changing grip positions can occur. In this way, a compression of the carotid bifurcation is a possible implication.

In healthy people, triggering a significant (lethal) cardiac reflex seems to be impossible, even with strong bilateral pressure on the carotid bifurcations [24,28]. In pressure experiments on about 8000 subjects, aged 15–95 years, no death occurred [21]. Also, in animal experiments, a lethal irritation of the carotid sinus was impossible [27]. In martial arts, blows directed to the lateral neck are used for a temporary knock-out of the opponent induced by syncopal events, but events of death have not been reported [8]. In medical practice, the cardiac reflex by massage or pressure on the carotid bifurcation is used to decrease high heart frequencies, but it also occurs unintentionally (e.g. in moderate pressure on the carotid artery after accidental carotid puncture).

Studies have shown that heart rate and blood pressure are affected by pressure on the carotid bifurcation only under certain conditions [14,17,28]. In a few cases, ventricular fibrillation was induced [2,15,19], but these cases exhibited increased hypersensitivity of the carotid sinus due to pre-existing cardiac arrhythmia and digitalis medication. An increase in carotid sinus activity (hypersensitive carotid sinus) occurs from the age of 40; from the age of 60 it occurs also with asystolia [5,14,17,20,28,35]. The reason for the increased carotid sinus activity is the age-dependent development of atherosclerosis of the carotid vessel wall. With pressure on the carotid bifurcation, the baroreceptors in the adventitia of the internal carotids are pushed against the plaques of the media. In this way, atherosclerotic alterations and diseases that promote atherosclerosis (hypertension, diabetes mellitus) lead to a hypersensitive carotid sinus. Space-consuming tumours of the head and neck [9], which press on the carotid bifurcation, as well as icteric disease [14] and medication (insulin, digitalis) can also increase the reflex activity of the carotid sinus [23]. In contrast, alcohol may reduce the sensitivity [11,12]. The susceptibility of the carotid sinus is of considerable importance. In a study with 3500 people,

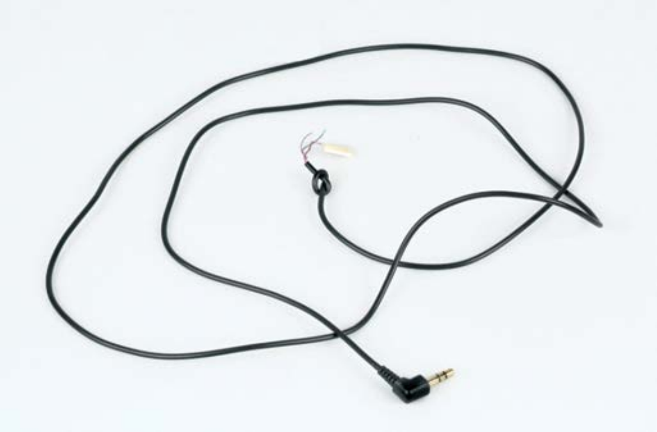

**Figure 25.8** Results study group: 35-year-old female; ligature strangulation with supralaryngeal ligature mark; autopsy findings: cyanosis, petechial haemorrhages, pharyngeal petechial haemorrhages, mild cerebral oedema, heart weight 300 g; cable tie used for ligature strangulation.

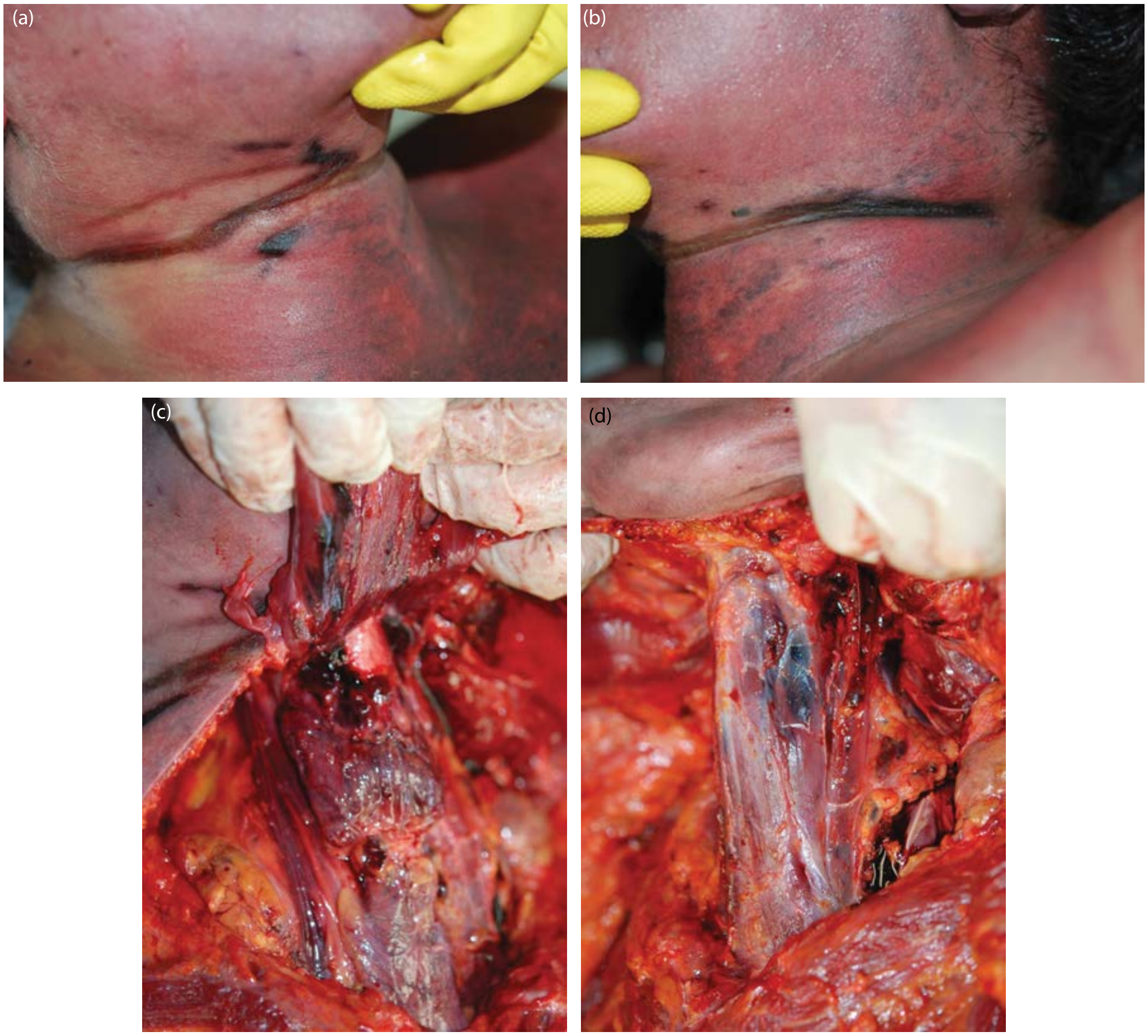

**Figure 25.9** Results study group: Ligature mark on the left (a) and right side (b) of the neck with haemorrhages into the tissue (c), (d).

Franke [14] revealed that a hypersensitive carotid sinus was present in 9 per cent of all cases. During autopsy, known pre-existing conditions and findings may be indicative for a hypersensitive carotid sinus. In our study group no considerable atherosclerosis or diseases were present and there was no evidence of hypersensitive carotid. Sigler reports on the occurrence of reflex-based death in the case of heart disease [32].

If it is possible that a lethal reflex death has occurred, many factors have to be considered. These include the history (e.g. witness statements), macroscopic and microscopic findings of violence against the neck (e.g. haemorrhage) as well as general macroscopic and microscopic findings (cyanosis, petechiae, haemorrhagic pulmonary oedema, acute pulmonary emphysema and cerebral oedema) including signs of prolonged circulation and hypoxia [31,33].

In the cases presented here, histories that pointed towards fast death (sudden collapse) were not present. Most of the cases were suicides, committed alone (sudden unconsciousness is normal for hanging) with mild trauma of the neck. In cases of hanging, haemorrhagic lesions of the neck tissue are typically not seen. However, the rope above the larynx can lead to pressure on the carotid bifurcation.

In all of the presented cases of neck trauma, signs of vital reactions and longer preserved cardiocirculatory activity (muscular haemorrhage, signs of venous stasis of the head or histopathological vital reactions) were present. An acute and fast cardiac reflex death was out of the question.

The histological examination of the carotid bifurcations with regard to the histomorphological correlate of tissue trauma (haemorrhage) did not reveal significant results. In only one case (case 20) was an evidential sign of direct trauma of the carotid bifurcation found (Figure 25.7). In this case of ligature strangulation (Figures 25.8 and 25.9), the histological examination of the carotid bifurcations revealed haemorrhage in the tissue outside the vessel wall and carotid body. Haemorrhage in the carotid body could not be detected. Due to swelling and lividity of the face, petechiae and haemorrhage in the musculature of the neck as a sign of preserved blood circulation, a reflexively quick cardiac arrest had to be excluded.

Sigrist et al. [33] reported on seven deaths resulting from massive impact to the neck (e.g. kick of a cow against the neck, karate hand coup against the neck or jammed neck). Their histological examinations found injured vessel walls of the internal carotid artery and haemorrhage into the reflex zones. Indications of fast cardiocirculatory failure (mild haemorrhage of the traumatized tissue, mild vital reaction) without relevant pre-existing heart disease were present. The cases reported by Sigrist et al., with clear findings of traumatized reflex zones and a background of acute severe violence against the neck, are in contrast with the presented cases, in which haemorrhage in the reflex zones was not detected due to comparatively mild trauma of the neck and carotid arteries.

Lethal cardiac reflex could also occur with delay, after already developed vital reactions (signs of venous stasis, especially in case of suffocation or massive haemorrhage) in case of slowly bleeding with gradually increasing pressure and persistent stimulation of the nerve fibres, while otherwise the absence of the trigger mechanism the cardiovascular activity quickly will be re-regulated. This effect is seen in Schollmeyer's report on a case of the reflex death of a man who was hit on the left side of the neck with an umbrella [30]. Sudden death occurred 4–5 minutes after the trauma. Histological examination of the carotid bifurcation revealed haemorrhage into the carotid body. Relevant pre-existing heart damage was present. A safe assumption of lethal cardiac reflex is admissible only where competing findings can be excluded.

One paper in the literature reports on a lethal carotid sinus syndrome that was probably due to an unusual complication after puncture of the carotid artery during positioning of a Shaldon catheter [9]. A 39-year-old woman was brought to hospital after acute renal failure following renal transplantation. Acute dialysis was planned: the right jugular vein should be punctured by a Shaldon catheter. However, the right common carotid artery was punctured. Bleeding into the soft tissue started immediately. After sudden and unexpected cardiac arrest, resuscitation was started but intubation failed. Stenosis of the trachea due to haematoma following the puncture was discussed as cause of death as well as thromboembolism and early septic process. Autopsy revealed an extensive retropharyngeal haematoma but no stenosis of the upper airways. Histological investigations showed extensive bleeding surrounding the sinus caroticum with subcapsular haemorrhages. The sudden onset of the cardiac arrest could have been initiated by a haemorrhage-induced irritation of the sinus caroticum.

## Conclusion

There is general agreement that the sinus reflex can occur and can rarely cause death, but it seems to play no significant role in healthy children, adolescents and young adults.

Without severe trauma of the neck, significant tissue trauma of the carotid body and carotid sinus can obviously not be expected in histological examination. Without history of rapid death, signs of fast failure of the cardiovascular system and macro- and histomorphological correlates of neck trauma (haemorrhage) within the chemoreceptor and especially pressoreceptors, the discussion of a possible reflex death is not appropriate and not reliably confirmable. A delayed carotid sinus reflex could occur due to persisting or increasing pressure on the reflex zones, but verification and chronological classification could be difficult or even impossible.

The question of whether a brief gripping of the neck without any signs of injury could cause death by the carotid sinus reflex, and if consequently every grasp to the neck could be suggestive of violence dangerous to life, was investigated some years ago in a bibliographical study. After evaluating many reports from clinical and forensic cases, the authors concluded that most case histories consist of only a brief description without any further explanation about details or results of postmortem examination. According to the literature study, sudden death after a short grip to the neck seems to be extremely improbable in a 'normal case' and should be taken into account only when special preconditions are present in the deceased [21].

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# 26 Bolus death

*Wolfgang Keil*

## Definition

Fatalities caused by obstruction of the larynx, also known as ‘bolus death’ after the Latin for ‘morsel’ or ‘lump’, are associated with a reflex cardiac arrest. The bolus material lodged in the throat triggers the reflex by impacting on the larynx. Virtually every case is caused by particles of food. Although the airways are obstructed, this does not constitute asphyxiation in pathophysiological terms. The food bolus (in rare cases other foreign bodies) becomes lodged in the laryngeal inlet due to a dysfunction of the swallowing process [6]. Due to the impaction of the larynx, the cough reflex mechanism is ineffective because the required air cannot be inhaled. The pressure irritates the parasympathetic nerve plexus of the mucous membrane at the inlet of the larynx and the pharynx. As a reflex, the vagal impulses lower both the heart rate and the stroke intensity. Cardiac conduction and irritability are reduced. Sudden cardiac arrest may follow, proving fatal in most cases. As early as the beginning of the last century Kolisko [5] put forward this explanation. Nevertheless, asphyxiation was later considered possible again [4]. Eye witnesses report a loss of consciousness with no precursory symptoms of respiratory distress, which supports the reflex theory.

## Frequency/occurrence

According to Patzelt’s data [7], forensic pathological case studies show a frequency of deaths due to laryngeal obstruction of approximately 0.5 per cent. In a recent study by Blaas et al. [2], the frequency of such cases is 0.18 per cent. Men are affected more frequently than women. It is reasonable to assume that a large number of such fatalities go unreported because doctors wrongly diagnose the subject’s sudden, silent collapse as coronary cardiac arrest. It used to be thought that the bolus mechanism could only be triggered by a relatively solid obstruction. Thus, chunks of meat (Figure 26.1) and sausage were predominantly identified as bolus. In individual cases, objects such as coins, dentures and layers of gauze used in oral hygiene were found in the larynx. A study by Berzlanovich et al. [1] showed that fatalities among the elderly may also be caused by soft, impacted food boluses (Figure 26.2). It has long been established that alcohol has an activating effect on the parasympathetic nervous system and an inhibitory effect on the sympathetic system. Thus, it stands to reason that a relatively large proportion of subjects are alcoholized during the bolus event [7].

## Classification of the circumstances

Approximately 70 per cent of bolus fatalities take place in the home, with the remainder mainly in restaurants, old people’s homes, care facilities and hospitals [1]. A link can generally be identified between the onset of death and the ingestion of food. Cases have been reported in kiosks, fast food restaurants and canteens. Geriatric patients or those with neurological disorders are particularly vulnerable as they are more likely to suffer from dysphagia [6]. In these categories of patient, sudden deaths while eating must always indicate the possibility of a bolus mechanism. The hurried feeding of elderly or handicapped patients evidently contributes to this type of incident. In most cases there are no indications of any vomiting as may be repeatedly observed in the aspiration of chyme. Occasionally, the circumstances of the demise or the anamnesis may provide evidence of alcoholization.

## Pathomorphology

### External findings

**Petechiae** These occur only very rarely in the conjunctivae or not at all.

### Internal findings

**Site of the bolus** This is located in the larynx and/or in the lowest section of the pharynx. As a rule, the laryngeal inlet is completely obstructed. Bolus death is evidenced by the presence of the foreign body. It should be noted that the bolus material may have been removed as a result of resuscitation measures or shifted during intubation.

**Stomach contents** In approximately two-thirds of cases, undigested food particles are found in the stomach that correspond to the bolus material and point to the ingestion of food immediately before the event. In one-third of subjects, the stomach is empty, i.e. the first mouthful proved fatal.

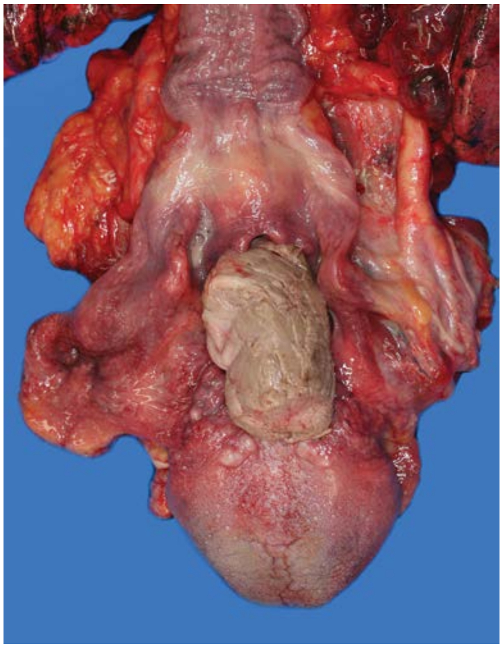

**Figure 26.1** Bolus death. A compressed piece of meat in the larynx and in the lowest section of the pharynx.

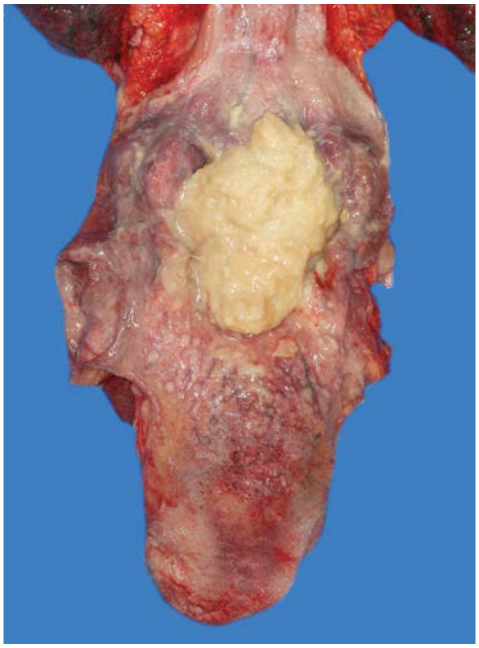

**Figure 26.2** Bolus death. Pressed potatoes in the larynx and in the lowest section of the pharynx.

**General findings** Uncoagulated blood and acute hyperaemia of the internal organs are often found.

## Additional examinations

**Blood alcohol test** The results frequently indicate medium to high concentrations.

**Chemical–toxicological analyses** These may lead to the detection of centrally acting drugs, which evidently also contribute to such fatalities.

## ■ Differentiation between suicide, homicide, accident and natural death

**Suicide** In principle, a bolus mechanism as a method of suicide would appear to be possible. Forster and Schulz [3] documented a case of self-inflicted gagging by a schizophrenic subject, which evidently triggered a bolus mechanism.

**Homicide** If elderly or handicapped patients with dysphagia suffer a bolus death after being wrongly given foods that have not been sufficiently broken down, it could be questioned whether this is criminal assault resulting in death. The same applies to the hurried feeding of such patients.

**Natural death** Since obstructions of the larynx are predominantly due to varying causes of dysphagia, a natural death may be assumed in many cases.

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# 27 Drowning

*Philippe Lunetta*

## Definitions

### Drowning

The World Health Organization (WHO) adopted a consensus definition elaborated in 2002 by a working group at the World Congress on Drowning (Amsterdam, Netherlands) which defines 'drowning' as the 'process of experiencing respiratory impairment from submersion/immersion in liquid' that can result in death, morbidity or no morbidity [150]. The terms 'submersion' and 'immersion' indicate the position of a corpse that respectively dips under the surface of a liquid or partially under respectively. Drowning can also occur when the face and external airway orifices alone dip under a liquid (Figure 27.1); a layer of liquid as shallow as 15–20 cm can cause drowning, generally in infants or in individuals who suffer seizures or are incapacitated by another medical condition or under the influence of alcohol or drugs.

Standardized definitions and uniform terminology on drowning are crucial to addressing all prevention, rescue and treatment issues [63]. Despite significant improvements, peer-reviewed scientific contributions still include non-uniform definitions and terminology, such as 'wet drowning', 'dry drowning', 'secondary drowning' and 'near drowning' [109,126,130]. To better comply with the WHO definition, 'near drowning' could be replaced with the term 'non-fatal drowning'. Contrary to the WHO definition, in forensic medicine the term 'drowning' implies a fatal outcome, drowning being mostly defined as 'death by penetration of liquid into the airways' or as 'death caused by submersion in a liquid' [29,46,49]. However, medicolegal expertise on drowning-related incidents is not limited to fatal cases but increasingly also covers non-fatal incidents in matters such as aquatic safety and professional medical liability [97]. Standardized definitions and uniform terminology therefore need implementation also in forensic medicine, and effort made to fill the gap between the forensic and the WHO definitions of drowning [18,155] and across English- and German-speaking countries.

### Drowning media

The 'drowning media' consists of any liquid substance which penetrates into the airways from the surrounding environment. Drowning does not include asphyxia caused by aspiration of the victim's own liquids such as gastric contents, blood, or amniotic liquid, for which the term 'choking' remains more appropriate.

Drowning media should not be characterized only in terms of salt-, brackish or fresh water, but also in relation to the tonicity of body liquids (see 'Pathophysiology'). For instance, as salinity of the Baltic Sea near the coast is lower (3–6 parts per thousand) than that of human body liquids (9 parts per thousand), in Finland most seawater drowning occurs in a hypotonic environment [74]. Conversely, drowning in hypertonic media may occur in inland water such as watercourses draining over-fertilized fields [25].

Chemical or biological properties of the drowning media, other than salinity, can prove clinically relevant. Drowning media containing bacterial pathogens and other microorganisms may cause – eventually in combination with aspiration of gastric contents and endogenous bacteria – fulminant or delayed pneumonia [38,120,136]. Drowning in ponds and stagnant water can result in invasive pulmonary aspergillosis whereas drowning water containing chemical pollutants or cleaning substances can result in chemical pneumonia.

Drowning may also occur in atypical liquid media and settings, including wine, and septic or manure containers. Drowning in beer, gasoline, oil, bitumen, paraffin wax and sugar syrup has been reported; in these incidents, individual properties of the medium and its environment can worsen the pathophysiological effects of liquid aspiration.

## Epidemiology

Drowning is the third leading cause of unintentional injury death worldwide, accounting for 7 per cent of all injury-related deaths. An estimated 370 000 persons die every year as a result of drowning [159,160,161]; among survivors, a significant percentage may sustain moderate or severe neurological sequelae.

Drowning rates vary widely among continents and regions. More than 90 per cent of all fatal drowning occurs in low- and middle-income countries (LMICs). The Western Pacific and South-East Asia WHO regions account for more than 50 per cent of the global mortality from drowning. Drowning mortality rates are highest in the WHO African regions and are 15–20 times higher than in some high-income countries (HICs) such as the

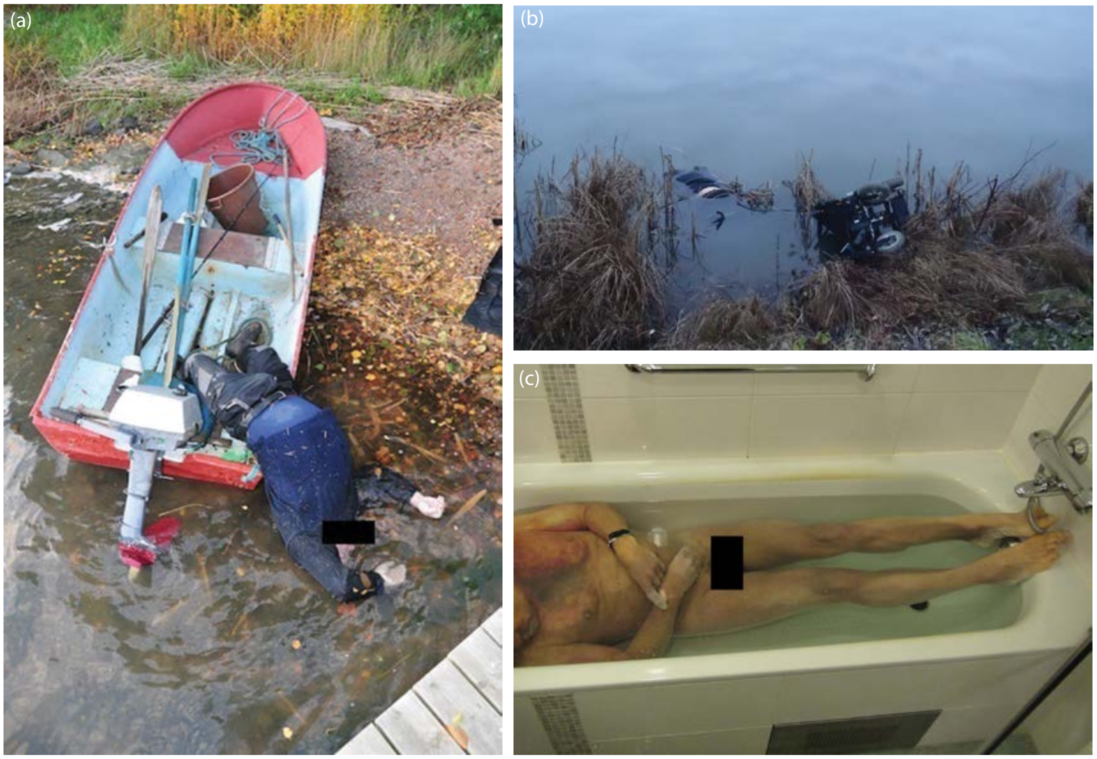


**Figure 27.1** Victims of drowning in shallow water. In (a) the external airways orifices and only part of the rest of the head dip under the water surface (b) pond and (c) bathtub.

UK and Germany [159,160]. In most European countries, unintentional drowning rates are lower than in LMICs, but differences exist among individual countries and regions. Baltic countries and some eastern European countries such as Poland have much higher mortality rates by drowning than western European countries [161].

The age distribution of unintentional drownings varies widely between countries with exceedingly high rates among children and adolescents, especially in LMICs, and a relatively higher proportion of drowning among adults in some high-income countries. Worldwide, the mortality rate by drowning in males is twice as high as in females.

The global WHO estimate of drowning deaths underrepresents the actual drowning figures. Collection of epidemiological data on drowning and cross-country comparisons based on WHO data are hampered by differing record-keeping systems and by differences in coding practices. Many LMICs collect no information or have incomplete recording of data on water-related mortality. Studies based on WHO standard data do not generally include boating-related accidental drowning, drowning from natural disasters, and land-traffic-related drowning [83,84,86,159].

The mortality rates of suicide by drowning vary widely between countries, for example from 0.1–0.2 per 100 000 population in the USA to 0.7–0.9 per 100 000 in northern Europe. The proportion of suicide by drowning among overall suicide ranges from approximately 1 per cent in the USA to 15–20 per cent in Ireland. A survey conducted in 16 European countries showed that drowning represented 3 per cent of all suicide methods in males and 7.7 per cent in females. When performing cross-country comparisons on suicides by drowning, one aspect which needs consideration is that a variable proportion of suicidal drowning may be classified among drownings of undetermined intent [76].

Data on homicide by drowning and drowning of undetermined intent are mostly limited to in-depth studies generally performed in HIC's selected populations or settings.

The WHO European regional office has recently made available [160,161] general data on drowning by manner of death (Table 27.1) and similar data are available for selected extra-European countries through the WHO Mortality Database and National Statistical Offices.

## Pathophysiology

The pathophysiology of drowning is complex and to a large extent still unknown. Moreover, the wide variety of scenarios in which drowning occurs means that in each drowning, some, but not necessarily all, pathophysiological mechanisms will take place. Drowning can occur in shallow water, with only the mouth and nostrils being under water, or in deep water of the open sea; in cold or in warm water while bathing or swimming or as a result

**Table 27.1 Drowning by manner of death in selected European countries and the US***

| | Accident[1] | | Suicide[2] | | Homicide[3] | | Undetermined[4] | |
|---|---|---|---|---|---|---|---|---|
| **Country** | ***n*** | ***n*/100 000** | ***n*** | ***n*/100 000** | ***n*** | ***n*/100 000** | ***n*** | ***n*/100 000** |
| Austria | 32 | 0.37 | 59 | 0.69 | 0 | – | 52 | 0.61 |
| Belgium | 67 | 0.60 | 130 | 1.16 | 2 | 0.02 | 37 | 0.33 |
| Bulgaria | 115 | 1.58 | 5 | 0.07 | 0 | – | 1 | 0.01 |
| Croatia | 79 | 1.86 | 24 | 0.57 | 0 | – | 7 | 0.17 |
| Cyprus | 15 | 1.74 | 1 | 0.12 | 0 | – | 0 | – |
| Czech Republic | 141 | 1.34 | 20 | 0.19 | 0 | – | 30 | 0.29 |
| Denmark | 48 | 0.86 | 34 | 0.61 | 0 | – | 5 | 0.09 |
| Estonia | 66 | 5.02 | 2 | 0.15 | 0 | – | 2 | 0.15 |
| Finland | 161 | 2.95 | 47 | 0.86 | 2 | 0.04 | 22 | 0.40 |
| France | 962 | 1.51 | 411 | 0.64 | 6 | 0.01 | 13 | 0.02 |
| Germany | 392 | 0.48 | 220 | 0.27 | 2 | 0.00 | 79 | 0.10 |
| Hungary | 128 | 1.30 | 45 | 0.46 | 1 | 0.01 | 51 | 0.52 |
| Ireland | 53 | 1.15 | 35 | 0.76 | 1 | 0.02 | 19 | 0.41 |
| Italy | 400 | 0.67 | 177 | 0.30 | 2 | 0.00 | 0 | – |
| Latvia | 183 | 9.18 | 4 | 0.20 | 1 | 0.05 | 10 | 0.50 |
| Lithuania | 229 | 7.81 | 9 | 0.31 | 1 | 0.03 | 31 | 1.06 |
| Luxembourg | 1 | 0.18 | 4 | 0.72 | 0 | – | 0 | – |
| Malta | 2 | 0.47 | 2 | 0.47 | 0 | – | 0 | – |
| The Netherlands | 94 | 0.56 | 105 | 0.62 | 0 | – | 4 | 0.02 |
| Poland | 757 | 1.99 | 12 | 0.03 | 0 | – | 138 | 0.36 |
| Portugal | 80 | 0.77 | 98 | 0.94 | 0 | – | 48 | 0.46 |
| Romania | 725 | 3.25 | 30 | 0.13 | 0 | – | 4 | 0.02 |
| Slovakia | 129 | 2.38 | 10 | 0.18 | 0 | 0 | 8 | 0.15 |
| Slovenia | 28 | 1.37 | 16 | 0.78 | 0 | 0 | 6 | 0.29 |
| Spain | 544 | 1.17 | 118 | 0.25 | 1 | 0.00 | 6 | 0.01 |
| Sweden | 128 | 1.32 | 65 | 0.67 | 0 | 0 | 26 | 0.27 |
| United Kingdom | 279 | 0.44 | 152 | 0.24 | 0 | 0 | 136 | 0.21 |
| USA | 3786 | 1.20 | 497 | 0.20 | 44 | 0.00 | 301 | 0.10 |

*Source:* WHO Detailed Mortality Database, 2017; Center for Disease Control and Prevention, National Center for Health Statistics, 2017.

[1] Accidental drownings, ICD-10: V90, V92, W69–W74.

[2] Suicide by drowning, ICD-10: X71.

[3] Homicide by drowning, ICD-10: X92.

[4] Drowning of undetermined intent, ICD-10: Y21.

* Data mostly 2014 and 2015; Greece: ICD-10 data not available.

of falling into water; or as a result of a land-traffic accident with complete or partial submersion of the vehicle.

In addition to the events that follow the aspiration of liquid into the airways, the pathophysiology of drowning includes protective autonomous responses (breath-holding, the diving response, acute hypothermia) and life-threatening responses (cold shock, autonomic conflict) of the body to immersion and submersion [11].

## Immersion

The effects of body immersion depend on water temperature, i.e. below thermoneutrality (temperature at which heat loss equals heat production: 35°C ± 0.5) or above it.

### Cold-water immersion

The majority of drowning incidents occur in water at a temperature below thermoneutrality. In this setting, the pathophysiological responses that may contribute to drowning are evoked by skin cooling (cold shock), cooling of superficial muscles and nerves, and cooling of deep body tissues (hypothermia: body temperature below 36.8–37.7°C).

At first, a fall into cold water evokes the cold-shock response that usually peaks during the first 30 seconds of immersion and occurs at water temperatures between 15°C and 10°C. Cold receptors in the subepidermal layer stimulated by the sudden decrease in skin temperature evokes – via the respiratory centre and spinal α-motoneurons innervating the intercostal muscles and diaphragm – a response that includes gasping and hyperventilation [11,143]. Gasping and hyperventilation lead to an inability to breath-hold and to an increase in risk of liquid inhalation.

As cold-water immersion (CWI) persists, the next tissues to cool are superficial nerves and muscles, especially in the arms. At muscle temperatures below 25°C, fatigue occurs, because low temperatures damage superficial muscle fibres, leaving a smaller number of fibres to produce the same force. Nerve cooling to temperatures between 5°C and 15°C for 1–15 minutes can lead to impairment similar to peripheral paralysis [14].

The signs and symptoms of deep body cooling (hypothermia) include shivering (36°C), confusion and disorientation (35°C), amnesia (34°C), cardiac arrhythmias (33°C), clouding of consciousness (33–30°C), loss of consciousness (30°C), ventricular fibrillation (28°C) and death (25°C). The temperatures in parenthesis represent only rough approximations. Clouding and loss of consciousness impede the victim's ability to take appropriate measure to avoid aspiration of liquid [11,13,105]. At temperatures below 28°C, cardiac arrest may occur, with most deaths occurring at body temperatures of 24–28°C.

Drowning at a water temperature below 5°C can have, especially in children, a better prognosis than in warm water. The high surface-area-to-weight ratio in children, together with aspiration of cold water, may cause a rapid deep tissue cooling below 30°C. In these conditions, reduced metabolism causes a decrease in $O_2$ consumption and in $CO_2$ production, which give a certain degree of cerebral protection against hypoxia. This explains the recovery of children with no sequelae after protracted submersion (up to 66 minutes) in cold water [14]. The reduced metabolism with low oxygen requirements results in hypoventilation, dilated pupils and lack of tendon reflexes, giving the appearance of death.

Another pathophysiological mechanism during immersion in cold water is the 'autonomic conflict' (Figure 27.2). CWI may activate two responses: the

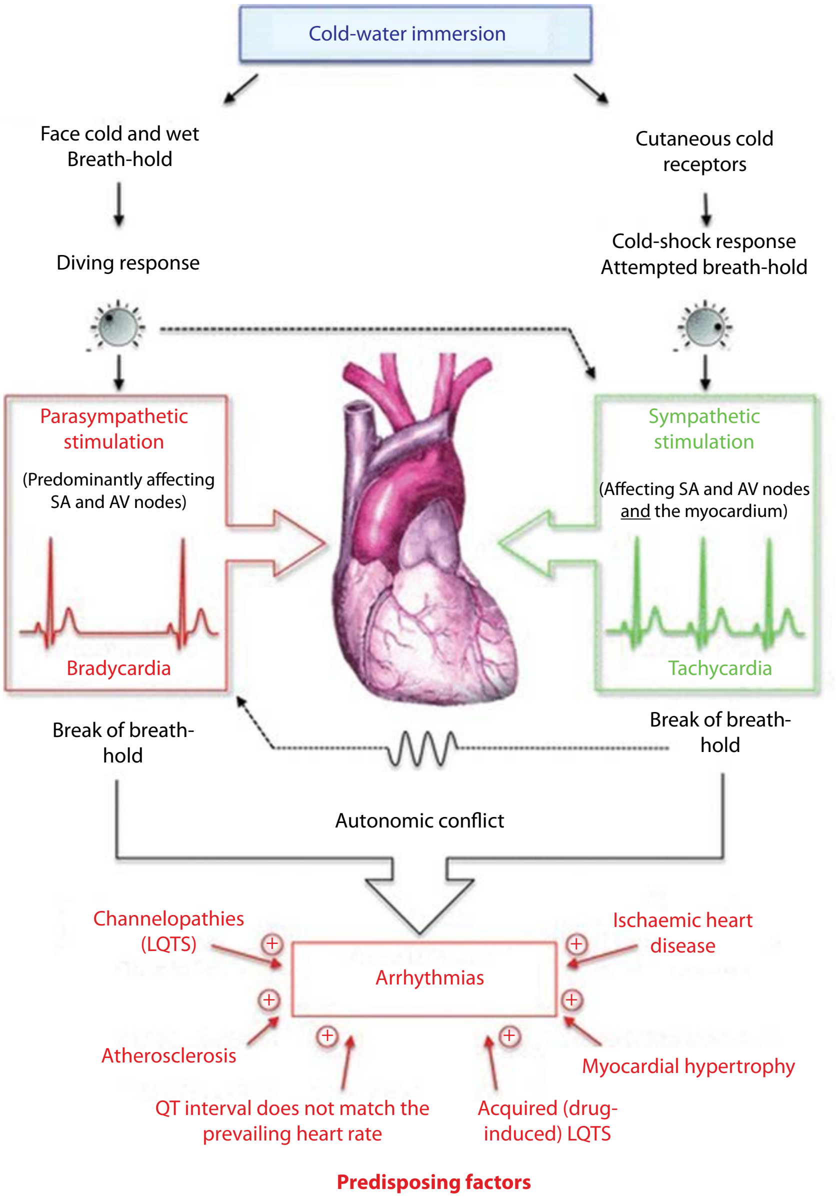

Cold-water immersion
Face cold and wet
Breath-hold
Diving response
Cutaneous cold receptors
Cold-shock response
Attempted breath-hold
Parasympathetic stimulation
(Predominantly affecting SA and AV nodes)
Bradycardia
Sympathetic stimulation
(Affecting SA and AV nodes and the myocardium)
Tachycardia
Break of breath-hold
Break of breath-hold
Autonomic conflict
Channelopathies (LQTS)
Ischaemic heart disease
Arrhythmias
Atherosclerosis
Myocardial hypertrophy
QT interval does not match the prevailing heart rate
Acquired (drug-induced) LQTS
Predisposing factors

**Figure 27.2** Effects of CWI: Diving response on facial immersion, cold-shock response on stimulation of thermoreceptors and autonomic conflict. (Reused with permission from Shattock MJ, Tipton MJ. *J Physiol* 2012;**590**:3219–3230.[131])

diving response on facial immersion that triggers a parasympathetically driven bradycardia (and splanchnic vasoconstriction with protective shunting of blood to the heart and brain) and this cold-shock response that activates a sympathetically driven tachycardia. These conflicting inputs to the heart can cause arrhythmias, especially at the break of breath-hold or within 10 seconds of its cessation. No conclusive evidence reveals whether in a healthy individual such arrhythmias, as well as those triggered by the cold-shock response, are by themselves life-threatening in healthy individuals. However, such arrhythmias can incapacitate the victims in water or eventually evolve into fatal arrhythmias in the presence of pre-existing cardiac disease(s) [10,11,131,142]).

### Hot-water immersion

The high incidence of fatalities in a hot-water tub, especially in Japan, suggests that hot-water immersion (HWI) may contribute to drowning [165]. Thermoregulation during HWI differs from that in hot ambient air mainly because, in a hot-water tub, evaporation of sweat is limited to the skin of the head and neck that are above water. The increase in skin temperature evokes – via cutaneous warm thermoreceptors and hypothalamic thermoregulatory centers – an efferent response consisting of vasodilatation, with tachycardia that may trigger ventricular arrhythmias [14,96,125,157]. This cardiovascular change occurs generally with protracted immersion at high temperature and may be life-threatening in subjects suffering pre-existing cardiac conditions [1,3,11,44].

## Submersion

Drowning has been described as a process that begins when the victim's airways lie beneath the surface of the liquid [63]. The 'instinctive drowning response' starts approximately 1 minute before the actual drowning process in deep water. It explains why drowning often occurs silently, with a motionless victim upright in the water unable to call and wave to attract help and then quietly disappearing under the surface. When a person is close to slipping under the water, he or she will breathe by rapidly opening the mouth but will not always have time to call for help, because the mouth is alternatively sinking and reappearing above the water surface. Likewise, these victims cannot wave for help because they extend arms laterally on the water's surface, rather than raising their arms vertically, a move that is instinctively perceived to increase or speed the risk of slipping below the surface.

The process of drowning comprises the following phases:

1. Voluntary breath-holding.
2. Reflex laryngospasm.
3. Laryngospasm resolution.
4. Penetration of liquid into the airways with gasping for air, dyspnoea and cough-like expiration.
5. Loss of consciousness.
6. Tonic–clonic seizures with persistent respiratory activity.
7. Terminal gasping and cardiocirculatory arrest.
8. Death.

Concerning breath-holding, at a comfortable air temperature, the breakpoint occurs after approximately 60–90 seconds, with alveolar $pCO_2$ ranging between 43 and 53 Torr. In warm water, the average breath-hold time is about 45 seconds, but some well-trained breath-hold divers can achieve several minutes before inhaling. In cold water, the breath-hold times may range from less than 10 seconds to more than 100 seconds. The maximum duration of breath-hold varies widely among individuals. Factors influencing breath-holding duration in air include metabolic rate, pre-breathing with hyperoxic or hypoxic gas mixtures, experience and psychological tolerance. In water, additional factors reducing the duration of breath-hold include alcohol intoxication, voluntary liquid aspiration in suicide and, in water below 15°C, the cold-shock response that can reduce breath-hold duration to just a few seconds [11,141].

Once they reach the breath-hold breaking point, the victims breathe, causing liquid to enter the airways and eventually be swallowed. Stimulation of the laryngeal mucosa by the liquid can lead to reflex protective laryngospasm to prevent foreign materials reaching the lower airways [103]. During laryngospasm, respiratory movements against the closed glottis may cause mechanical damage to the pulmonary alveoli. A certain agreement exists over the transient nature of laryngospasm. As arterial oxygen tension drops and hypoxia ensues in laryngeal muscles, laryngospasm will abate, followed by involuntary gasping and penetration of liquid into the airways [11].

## Organ effects

### Respiratory system

Once liquid penetrates into the airways, the lungs become the primary vulnerable target.

Both hypertonic and hypotonic liquid damages pulmonary surfactant and the alveolo-capillary barrier, with resulting ventilation/perfusion shift, hypoxaemia and hypercapnia, metabolic acidosis, cerebral anoxia and death. It is currently accepted that the main pathophysiological consequence of liquid aspiration is hypoxia [11,63,107].

Hypotonic liquid that is partially absorbed into the pulmonary circulation damages pulmonary surfactant, increases alveolar surface tension and reduces pulmonary compliance. The resultant atelectasis alters ventilation-to-perfusion ratios: blood perfusion occurs in non-ventilated areas and venous blood bypasses the lungs. Up to 70 per cent of the cardiac output may be shunted past perfused but unventilated alveoli. Hypertonic media draw liquid from the plasma into the alveoli, which also results in damage to the surfactant. One estimate is that 2.5 ml/kg of seawater may cause the pulmonary shunt fraction to increase by 75 per cent, and that as little as 1–3 ml/kg may reduce pulmonary compliance by 10 per cent to 40 per cent [48,80,99,100].

#### *Dry drowning*

Clinical and experimental data together with autopsy findings of apparent 'dry-lungs' have suggested that death in water can occur with no significant aspiration of liquid (dry drowning) by contact of the liquid with the upper airways that triggers mechanisms such as protracted laryngospasm or vagally mediated cardiac arrest [81]. The laryngospasm hypothesis has its rationale in the complex innervation of the upper airways and their reflexes to various stimuli [11]. No evidence exists, however, that prolonged laryngospasm persists until death, and it seems plausible that laryngospasm abates within minutes when arterial oxygen tension drops and hypoxia in laryngeal muscles ensues. Complete penetration of hypotonic liquid into the bloodstream after prolonged resuscitation has also been proposed to explain dry lungs, but this hypothesis contrasts with the observation of dry lungs also in non-resuscitated victims of seawater drowning.

The contribution of forensic pathology on the issue of dry drowning has been confusing. Dry lungs have been reported without any precise definition, often simply by using an arbitrary cut-off weight (1000 g), although no consensus exists for normal lung weight. However, one autopsy study based on 578 definitive drowning victims with no putrefaction changes has questioned the actual occurrence of dry lungs. That study revealed that almost all victims with apparently normal and low-weight lungs presented other changes associated with liquid penetration into the airways (external foam, frothy liquid in the airways; diatoms) (see 'Postmortem findings') [81].

The actual occurrence of dry drowning has been questioned during the past two decades, and a consensus now exists that the term 'dry drowning' should be discarded, as it represents a nonentity [63,81,98,85].

### Cardiovascular system

Cardiac dysfunction occurring during drowning is mainly secondary to changes in arterial oxygen tension and acid–base balance. Acute hypoxaemia results in catecholamine release, leading to transient tachycardia and hypertension, followed, as hypoxemia intensifies, by bradycardia and hypotension. Hypoxaemia and acidosis reduce myocardial contractility and lead to increased risk for arrhythmias. In addition to myocardial depression, systemic hypoxemia causes reflex pulmonary vasoconstriction and changes in capillary permeability, all of which worsen pulmonary oedema [80].

### Central nervous system

Brain death is the common final stage in the pathway of fatal drowning. Cerebral hypoxia causes unconsciousness usually within 3 minutes; if effective CPR is applied within 3 minutes from the beginning of submersion, the vast majority of victims will successfully be resuscitated with no significant sequelae; by the time 5 minutes have passed, although an effective heartbeat can return, the majority of victims will sustain permanent hypoxic damage [75]. However, a notable exception to these time frames exists, especially in cold water and for children, for whom successful resuscitation with no neurological sequelae has been reported even after more than 30 minutes of submersion [11].

### Electrolyte disturbances

Experimental studies on drowning conducted on dogs during the 1940s and 1950s by Swann and colleagues [167] suggested that changes in serum electrolyte concentrations caused by liquids of different osmolarity were central pathophysiological mechanisms leading to death by drowning. These experiments are still misleadingly cited in some forensic pathology textbooks. Briefly, it was estimated that, in fresh water, the penetration of hypotonic liquid into the circulation causes hypervolaemia, erythrocyte lysis with intravascular $K^+$ release, and ventricular fibrillation (VF). Conversely, in hypertonic media, death is delayed compared with in fresh water because no haemolysis and VF occur. Further studies beginning in the 1960s have disclosed the limited clinical importance of serum electrolyte disturbances in human drowning and have demonstrated that liquid redistribution within the body compartments rapidly restores normal blood volume and electrolyte concentrations [135].

Clinically significant electrolyte changes have been observable only in specific environments, for instance in the Dead Sea [71,163] and in polluted water [43]. In specific circumstances, such as protracted immersion while wearing a malfunctioning life-jacket, seawater ingestion directly causes hypernatraemia [37]. High serum sodium concentration (>145 mM) has been described also in some paediatric drownings [59]. Electrolyte disturbances are generally diagnosed in non-fatal drowning but not postmortem (PM). In the latter cases, what can be challenging is to disentangle the role of ingestion and aspiration, because the small intestine absorbs about 80 per cent of all ingested liquid, including sodium, via concentration gradients and complex molecular mechanisms.

## Postmortem diagnosis

Determination of the cause and manner of death (accident, suicide, homicide, natural) in bodies recovered from water requires a comprehensive approach based on assessment of environmental circumstances, critical evaluation of eyewitness accounts, review of the victim's individual and medical background, and thorough analysis of autopsy findings [74].

Unfortunately, in a number of countries and jurisdictions, the task to investigate a body found in water rests with a medical doctor or another authority who lacks any forensic or medicolegal training, and the cause of death as drowning is established without autopsy, solely on the basis that the body is found in water [89].

## Drowning vs other causes of death

Although drowning is most frequently the underlying cause of death, a range of other causes, mostly diagnosed only at autopsy, may be responsible for death in aquatic settings. The author (PL) has investigated bodies found in water in which the underlying cause of death was a distinct medical condition (intracerebral and subarachnoidal haemorrhage, acute myocardial infarct, cardiac tamponade) occurring either when the victim was on dry land or on board a vessel, causing a fall into water, or occurring while the victim was immersed in water. Lacking any circumstantial information, it can be impossible to establish whether such a fatal medical condition existed prior to the victim's entering the water or occurred during the immersion as a consequence of physical exertion during swimming or a struggle to avoid submersion. A severe injury sustained by the victim immediately before or during a fall into water or while immersed can also have been the sole cause of death. Similarly significant could be fatal injuries inflicted purposely on land and followed by disposal of the victim's body into water.

When autopsy reveals no unequivocal natural or traumatic cause(s) of death, pre-existing medical conditions as playing a role as the underlying cause of death in water is exceedingly difficult or impossible to assess. Pre-existing medical conditions, injuries and intoxication(s) can contribute to drowning by causing the victims to fall into water or by incapacitating them while in the water. Such triggering factors can result in the victim's aspiration of liquid, but it may be impossible to state whether this aspiration occurred during terminal gasping or whether liquid penetration alone caused death by drowning.

Similarly, life-threatening cardiac arrhythmias can prove the sole cause of death or, as benign arrhythmias, can contribute to drowning. However, as the role of arrhythmias cannot be demonstrated at autopsy, this distinction remains only speculative, except in very rare cases where victims of drowning are promptly rescued and ECG records exist showing the electrical activity of the victim's heart at the time of resuscitation. Moreover, if medical records mention a genetically determined or acquired arrhythmia, or if PM molecular investigations disclose a genetic mutation responsible for life-threatening arrhythmias (e.g. long-QT syndrome), no definite conclusion should be drawn as to their actual role in the event leading to death. In criminal cases, however, such arrhythmias may be used by the defendant to raise reasonable doubts about the actual cause of death and possibly counter an allegation of homicide by drowning.

Once the cause of death as drowning is determined, the manner of death (accident, suicide or homicide) must also be ascertained. Despite thorough PM investigation(s) and medicolegal autopsy, the manner of death can at times remain undetermined.

## Challenges and investigative steps

Multiple challenges arise during scene investigations related to bodies found in water: they are conducted either for the purpose of criminal investigations or for safety investigations. The volatile scene is usually wider and less defined than it is on land and can extend to an underwater environment.

Often the fatal events go unwitnessed, with the reporting person as the only individual to have seen the victim in water; or the site of corpse retrieval from water does not match the site of death; furthermore, a prolonged PM submersion time alters the original scene at the time of death. For example, among 2125 deaths in aquatic settings that occurred in southern Finland during the period 1987–2012, only 25.1 per cent were eye-witnessed, and in nearly 50 per cent of the cases PM submersion time was at least 24 hours. In the same series, among 492 victims of suicide by drowning, only 23.6 per cent left a suicide note [75] (Table 27.2). Attempting to fit the circumstances to evidence from witnesses or reporting persons can bias the initial investigation. This means that the questioning and cross-examination of involved parties and witnesses should seek possible discrepancies.

In addition to the technical and tactical investigations of the police themselves, crucial steps for evaluation of the cause(s) and manner of death in bodies found in water are:

1. Victim identification.
2. Evaluation of PM time of submersion.
3. Identification of actual site of death.
4. Assessment of environmental circumstances.
5. Assessment of victim's personal and medical background.
6. Critical analysis of PM findings.

Especially in criminal investigations, any forensic pathologist, police investigators and other professionals must cooperate closely during all these steps, but these actors should not rely exclusively on each other to detect any findings that may raise the suspicion of a homicide [89].

### Victim identification

Personal identification is crucial, as it allows linking a victim's personal and medical background to the time and scene of death, to circumstantial evidence and to autopsy findings.

**Table 27.2 Selected factors that may hamper the evaluation of cause and manner of death in bodies found in water (based on 2125 bodies found in water, investigated in southern Finland, 1987–2012) [74]**

| Factor | % |
|---|---|
| Lack of eye-witnesses | 74.9 |
| Accident | 65.1 |
| Suicide | 89.0 |
| Homicide | 56.5 |
| Undetermined | 94.7 |
| Natural | 77.8 |
| Sea | 72.8 |
| Lake | 69.1 |
| River | 69.9 |
| Swimming pool | 81.1 |
| Bathtub | 98.6 |
| Protracted PM submersion time (≥24 hours) | 45.3 |
| ≥7 days | 21.4 |
| ≥1 month | 9.6 |
| ≥3 months | 6.4 |
| ≥1 year | 0.4 |
| Lack of suicide notes | 74.4* |
| Lack of drowning-associated autopsy changes | |
| External foam | 81.5 |
| Frothy liquid in airways | 61.9 |
| Overdistension of lung | 61.1 |

* Among 492 suicides.

In drowning occurring in domestic settings or with immediate recovery of the corpse from water after a witnessed event, prompt identification is generally possible by means of visual characteristics and personal effects. However, identification may also be hampered during the early PM period, if the victim does not carry identity documents or other personal effects, or if water has damaged these or washed them away. Personal identification is more challenging when corpses have undergone advanced decomposition.

The common medicolegal criteria for identification also apply to bodies retrieved from water. Comparisons between antemortem (AM) data, if available, and PM findings for scars, tattoos, physical malformations, evidence of past surgery and application of prostheses may assist in identification. However, identification at times must be confirmed by DNA, odontological examination and fingerprinting; DNA from bones may at times be the only suitable method, for instance in a body dismembered or suffering extreme decomposition or both. Advanced maceration changes do not preclude identification by means of fingerprinting, as suitable ridges may be obtained by specific methods [67].

Mass disasters at sea involving natural events (flooding, tsunami), and aircraft and ship disasters, including those transporting clandestine immigrants, raise specific identification issues. Drifting of a body from one's country coast or along rivers, as well as bodies of foreign nationals or tourists may require international cooperation in obtaining AM data, dental charts and DNA from the putative victim or relatives. Such cases require the use of Interpol Disaster Victim Identification AM and PM forms [134].

## Postmortem submersion time

The time since death is important information for the police investigation to position the victim – and in criminal investigations also the suspect(s) – in a given place, to analyze their last moments, to search for witnesses and to evaluate the environmental circumstances (e.g. regarding sea or weather conditions) at the time of death.

Evaluation of time since death is not an issue in drownings that include reliable witness accounts, but depending on the setting, this is not necessarily the case. Early (hypostasis, rigor, cooling, skin maceration) and late (putrefaction, adipocere formation, skeletonization) PM changes may be useful in evaluating time since death, but what must be understood is that PM changes in aquatic environments evolve differently and more unpredictably than on dry land. Distinctive factors that can shape PM changes in aquatic settings are the position of the body (complete submersion vs immersion with partial exposure to air); movement of the body (floating, sinking, drifting); and environmental factors such as air and water temperature, water flow and bacterial content of the water. The latter factors can change over time and with drifting or other movement of the body in water. Moreover, evaluation of time since death in cases of homicide on land followed by disposal of the body in water, or vice versa in homicide by drowning followed by disposal on land, must take into account the chronology of PM changes in both the land and the aquatic setting.

German authors [90,91] have modelled charts in which they have tabulated PM changes detected at external and internal examination in terms of time and water temperature. The value of these charts in assessing the time since death is, however, limited to local or regional settings. Moreover, additional ambiguity when using the charts arises from the difficulties in objectively grading PM changes [112] and from the rapid decomposition that can occur during the brief interval between a corpse's recovery and its external examination at morgue or autopsy room (Figure 27.3).

### *Early postmortem changes*

*Skin maceration*, characterized by thickening, wrinkling and whitening of the skin, occurs first on the fingertips, palms and back of the hands, as well as on the toes, soles and backs of the feet, and in cases of prolonged submersion this can sometimes also be visible on the elbows and knees. Further exposure to water causes progressive loosening of the nails and skin peeling from the hands and feet in a 'glove and stocking fashion' (Figure 27.4).

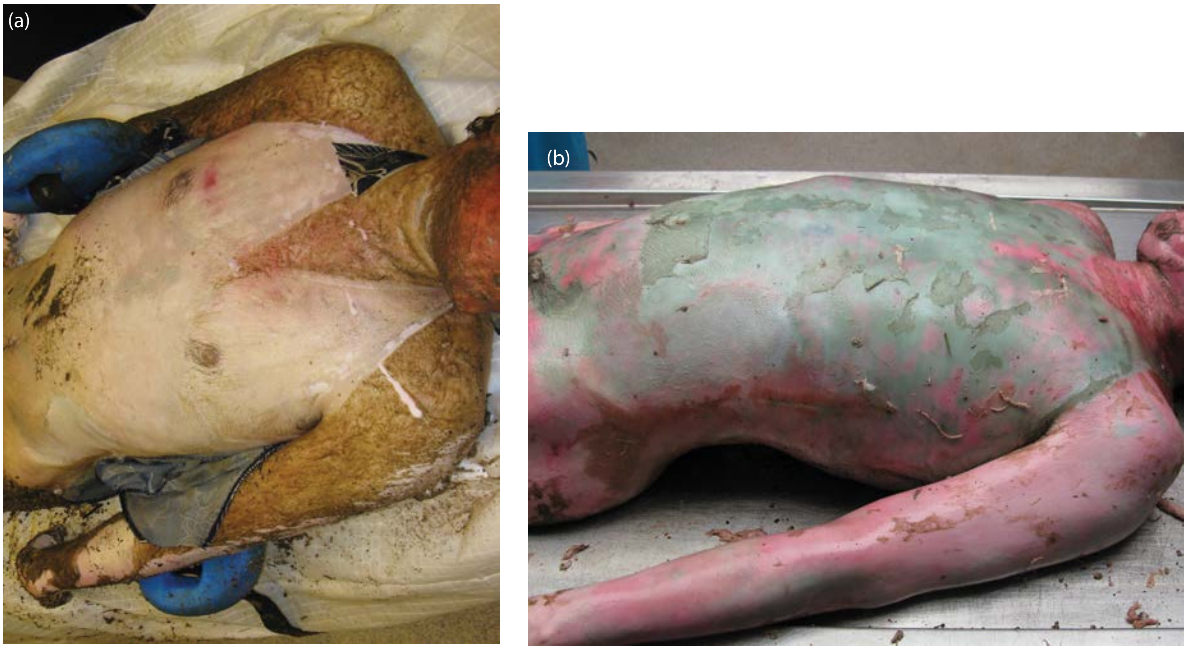
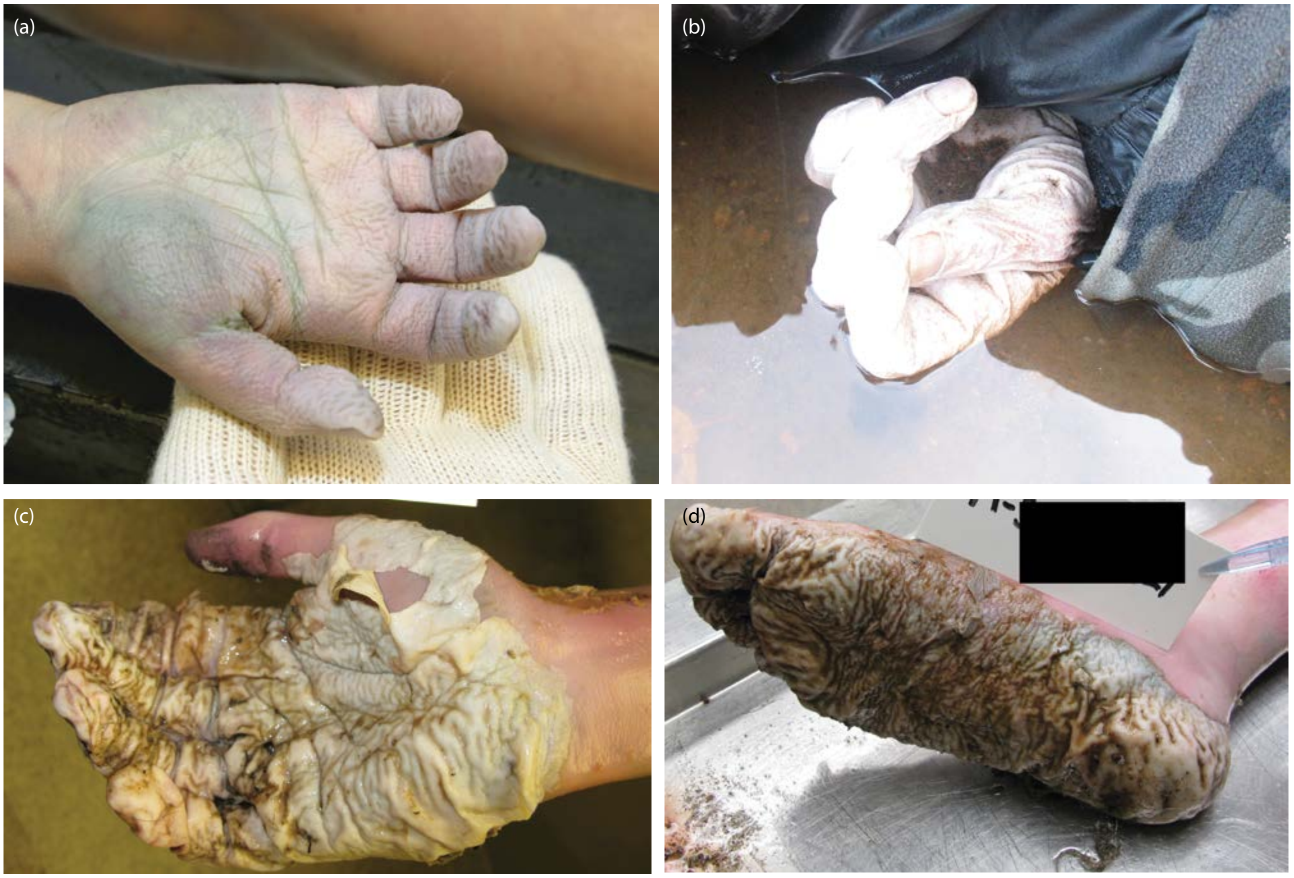


**Figure 27.3** External examination of a body found in water: (a) At the site of recovery; (b) at the morgue less than 24 hours later. Note the rapid progression of putrefactive changes which may be misleading if estimation of the time since death is based on the findings at the morgue alone.



[redacted]

**Figure 27.4** Different degrees of skin maceration in hands (a),(b),(c) and a foot (d). Different factors affect the timeline of maceration changes, the most important being the temperature of the water from which the corpse is retrieved. Gloves, footwear and socks are among other factors which may also influence this timeline. In (b) the palm of the hand contains mud and sand possibly grabbed by the victim from the bottom.

Conceivably, maceration is due to lack of the dynamic balance existing *in vivo* between liquid uptake and its reabsorption into the bloodstream, with continuous water input and formation of subepidermal liquid collections [156]. The chronology of maceration is somewhat unpredictable and depends primarily upon water temperature, but it is also influenced by gloves, shoes and socks worn by the victim. Roughly, in warm water the onset of maceration on the fingertips occurs within minutes, and skin peeling and nail loosening as early as in 3–4 days; in cold water, initial maceration changes can take up to 4–5 hours to manifest, and skin peeling and nail loosening may require 2–4 weeks.

In still water, as the corpse floats usually partially prone in a head- and leg-down position, *hypostases* are selectively located on the face, the upper part of the chest, in the thighs, calves and feet. However, hypostases can also appear posteriorly if the victim floats in a supine position with clothes forming air-collection spaces around the body. Any abundant adipose tissue in the breast and belly regions can also modify normal buoyancy. Conversely, in seawater and river settings, hypostasis due to the movements and rolling of the corpse in water can appear in any pattern, on any parts of the body. The chronology of hypostasis in water is comparable to that occurring on land; in cold water the pink-reddish colour of hypostases depends on exposure to low ambient temperatures. In victims of electrocution in a bathtub, hypostasis may present in a specific pattern, with the upper margin along the water surface line [15]. The thin and pale mark parallel to the water surface has been interpreted by some authors as a thermal phase-transition change independent of hypostasis, but it has also been described in cases involving no electrical contact [128,158]. At times, the teeth of a corpse in water may present with a pink discolouration (pink teeth), but this change is also observable on dry land [22]. It has been hypothesized that the head-down position that a corpse assumes while floating increases the venous pressure in the pulp, and after haemolysis enhanced by the moist environment, haemoglobin and its breakdown products move through the dentin.

Two main factors may influence *rigor mortis* in an aquatic environment. Low water temperature usually retards the development of rigor, whereas a victim's muscular activity while swimming or struggling before drowning and muscular contractions during drowning lead to earlier onset, stronger development and longer persistence of rigor compared to that on land. Grasping of seaweed and other marine material, especially from the bottom, may be a vital phenomenon but it may also occur PM during development of rigor mortis in the articulations of hands and fingers.

The *cooling* rate of a body in water is faster than on land since the thermal conductivity of water is more than 20 times higher than that of air. One estimation is that a naked corpse in cold water can cool approximately twice as fast as does a body on land [46], and once ashore, cooling is even more rapid due to liquid evaporation from the skin. This rapid cooling limits the use of rectal temperature to estimate the time since death to the first hours of submersion. German investigators have addressed the issue of body cooling in aquatic settings and discussed the effects of water temperature, current and the victim's clothing on the constants of Marshall and Hoare's formula [57,58].

### Late postmortem changes

The decomposition of a corpse in water is influenced by factors such as its position (floating or lying on the bottom), water temperature and bacterial content, and the victim's injuries. When a body is floating or partially washed ashore, body parts remaining immersed may disclose PM changes different from those exposed to air due to different air and water temperatures and may disclose different patterns of PM injuries produced by marine and terrestrial scavengers. A body sinking and its late resurfacing as well as its drifting on the surface expose a corpse to different environmental conditions over time and thus can modify the progress of PM decomposition.

*Chromatic skin discoloration* develops in water more slowly than in air because the cooler water temperature inhibits bacterial and insect activities. At water temperatures of 5–6°C or less, PM chromatic skin changes fail to develop, even after weeks, whereas at 15–20°C or higher temperatures, these can appear within a few days and be rapidly associated with large areas of epidermal detachment and the loosening of hair and nails. At this time, algal colonization may give exposed tissue a green or black discolouration.

*Adipocere* is a waxy greyish-white or tan decomposition product formed by anaerobic bacterial hydrolysis and hydrogenation of fat tissues [137] that can develop PM on a body's surface and on internal organs. In a submerged body, it occurs more frequently in the absence of oxygen and a wet environment such as the muddy bottom of a lake. Adipocere usually appears after a few months of submersion, but early formation has been observed occurring even after 3–4 weeks [32,65,132].

*During late decomposition,* scavengers' feeding activity causes PM artefacts in the corpse which can hamper identification, mimic or modify AM injuries and damage internal organs such as the heart and lungs. Scavengers quicken the course of skeletonization and disarticulation but can colonize the body even before the onset of putrefactive changes [35,53,152].

A recent study stressed the difficulties in evaluating time since death in water based on late PM decomposition changes [24]. During the course of skeletonization, joint disarticulation is enhanced by marine and fluvial currents and movement of the corpse in water during drifting. Disarticulation occurs first distally in the limbs due to higher torsion forces, while sinovial joints such as the shoulder are disconnected before the fibrous intervertebral ones. Studies on waterborne cadavers show a disarticulation sequence starting from the bones of the hands and wrists followed by bones of the ankles, mandible, cranium, legs and arms. Often the most buoyant disarticulated portion is a foot enclosed in a shoe that on occasion is found on a beach and traced to a drowning victim [85].

### Site of death vs site of corpse retrieval: Cadaver buoyancy and drifting

The site of death can be identical to or close to the place where the body is retrieved from water or it can be a remote aquatic setting. In homicides followed by disposal of the body into water, the site of death can be a distant location on dry land, and vice versa in homicidal drowning with disposal of the corpse on land.

Uncovering the site of death allows experts to link the case to a specific environment, to verify the movements of the victim and, in criminal investigation, of the suspect(s), to search for witnesses, and to perform a scene investigation, eventually extended underwater.

The basic notion of cadaver buoyancy and drifting is central for forensic investigation to locate the site of death and the site of retrieval of a corpse that has entered a body of water. Computerized models for simulating currents may serve in locating a corpse based on drowning site, to locate the site of entrance into water based on recovery site, or to set geographic and time limits for the search and thus for the PM interval. Studies on drifting of bodies using dummies have, however, highlighted that ‘each river must be examined in its own case’ [19,20,36].

Buoyancy and sinking of corpses follow basic laws of physics. A human body immersed in a liquid is under the action of two opposite forces: buoyancy force, exerted upward by the weight of the liquid displaced, and body weight, which exerts a downward force (Archimedes’ principle). When the body’s specific gravity is lower than that of the liquid, the corpse floats, with the reverse being true when body density is higher than that of the liquid. The specific gravity of a healthy adult (approximately 1.035–1.110) is slightly higher, but matches within a few per cent that of seawater on the surface (1.020–1.030), whereas the specific gravity of fresh water is 2–3 per cent lower than that of seawater. Hence, in principle, corpses have a higher specific gravity than seawater and fresh water and thus all should sink after death. Common experience is, however, that immediately after death some cadavers tend to float and others sink. Indeed, corpse buoyancy is enhanced by factors such as clothes that can trap air around the body, high content of adipose tissue (specific gravity 0.94) and high volume of air in the lungs [68]. A study on living male volunteers revealed that, at functional residual capacity (a situation similar to a recently dead body), 69 per cent could float in seawater and only 7 per cent in fresh water [33]. The low specific gravity of the trunk compared to that of the limbs also explains why a floating corpse will most often assume a head-down position with head, arms and legs towards the bottom; however, several factors– for instance clothes and high content of adipose tissue in the breasts and belly – explain why a corpse can also float face upwards in a semi-supine position (Figure 27.5).

In the case that the corpse sinks, as PM putrefactive gases develop, the corpse’s specific gravity decreases, causing it to resurface and float. In warm climates, rapid production of PM gases may cause the body to resurface within a few days after death. Putrefactive gases may cause a body to ascend to the surface even if it is secured to a heavy weight. In cold environments (high latitudes, great depths, mountain lakes) PM gas production can be markedly delayed or lacking so that the corpse can lie at the bottom for up to several weeks or months or, in some cases, not resurface at all [89].

In lacustrine environments, corpses are usually retrieved in still water close to the drowning location, on the bottom or, less often, floating. In fluvial environments, the corpse usually sinks, moves along the bottom, and resurfaces. Surface drifting depends on primary currents which flow downstream in the middle of the river and on secondary currents flowing toward the banks [89]. In marine environments, the drifting of a corpse is more marked at the surface than on the bottom. Drifting on the surface can begin immediately after death or occur later once the corpse has resurfaced as a result of PM gas production. Corpses floating can drift on the sea surface hundreds of kilometres from the point of entry into water in a relatively short period of time, depending on factors such as currents, tides, waves and winds.

### Environmental circumstances

Environmental factors are important from a forensic and preventive perspective to reconstruct all the events leading to drowning. Among the risk factors that should be considered are adverse climatic conditions, low-temperature water, environment unfamiliar to the victim, access to an unfenced body of water, inadequate level of parental or professional supervision, low compliance with vessel safety rules, and a malfunctioning life jacket. Assessment of environmental circumstances and individual factors leading to death in aquatic environments may require multidisciplinary investigation (expertise in crime investigation, forensic pathology, swimming instruction, life-saving, sea rescue, behavioural sciences) with a higher potential for accuracy than is contributed by police or medicolegal investigations alone [87].

### Victim’s individual and medical background

The assessment of a victim’s background ranges from review of criminal records to assessment of pre-existing medical or psychiatric conditions and medication. Furthermore, level of swimming proficiency, history of risk-taking behaviour, and alcohol and drug abuse can assist in identifying the events contributing to the actual drowning episode. Epilepsy is the single medical condition most studied and positively linked to drowning [8,27,42,106,124]. From

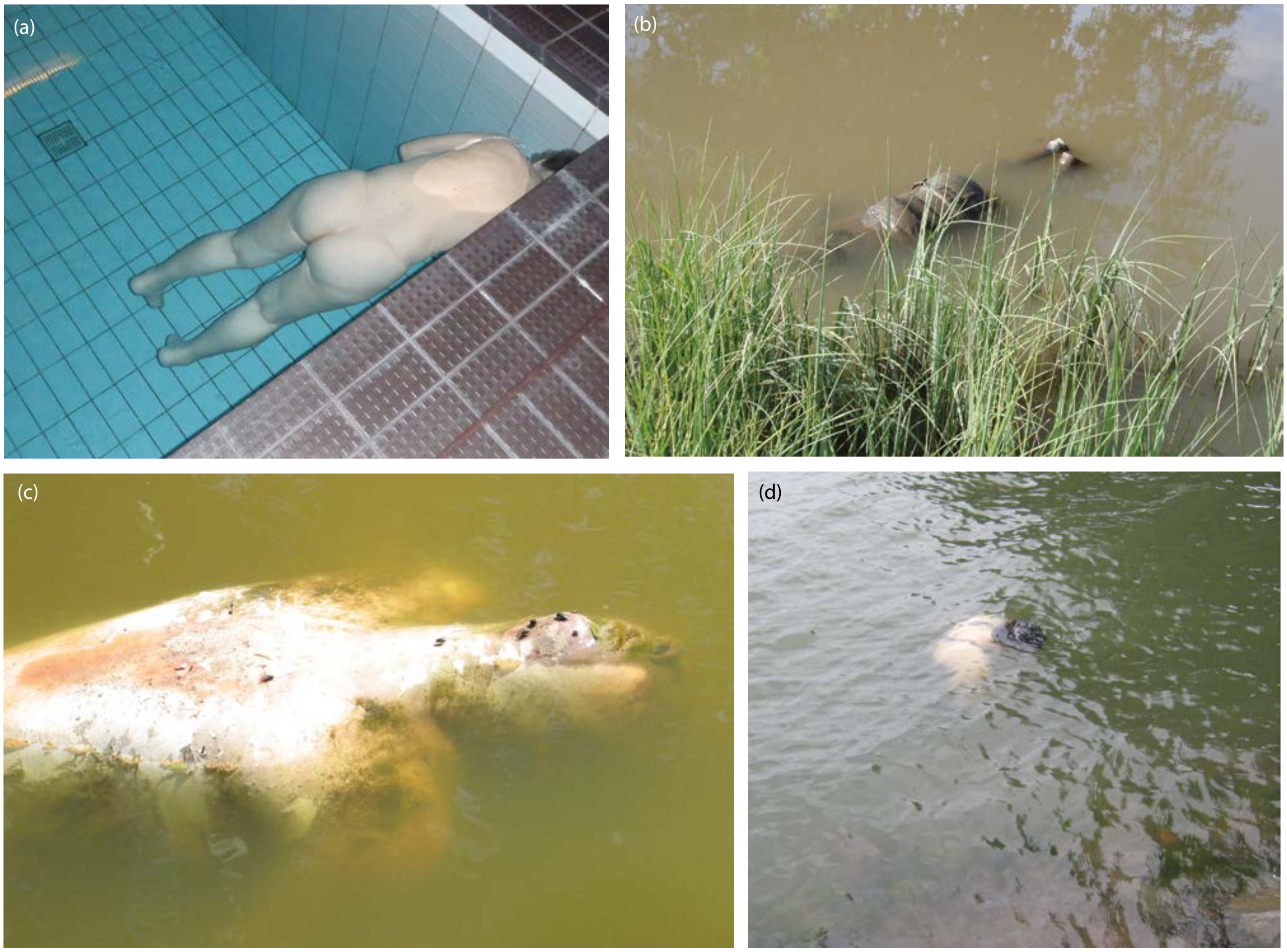


**Figure 27.5** Buoyancy and different postures a floating body may assume on the water surface. (a),(b) Typical head-down position with arms and legs towards the bottom, the trunk being the most buoyant part of the body. In (a) the victim wore around the trunk a bag containing heavy stones; (c) body floating in a supine position; (d) body floating in an upright, almost vertical position. Among the factors enhancing buoyancy and posture of the body are clothes that may trap air around the body, high content of adipose tissue and high salinity of the water. Some corpses may sink immediately after death and later become buoyant and resurface due to the late formation of postmortem gases.

a medicolegal standpoint, epilepsy should be selected as the cause of death or as a contributing factor to death only when death was preceded by seizures.

### Bathtub deaths

The investigation of bathtub deaths exemplifies well the challenges related to diagnosis of cause and manner of death in bodies found in water. In bathtub deaths, a range of causes and contributing factors can determine death at any age as a consequence of medical condition, accident, suicide or homicide. In addition to drowning, electrocution, carbon monoxide intoxication, drug overdose, sharp or blunt violence, or strangulation can be among the causes of death. Drowning can result from diminished consciousness or unconsciousness caused by disease (epileptic seizures, cardiovascular conditions) or, in both accident and suicide, by alcohol and drugs. All these factors may result in the victim's slipping beneath the water surface. The diagnosis of natural death in a bathtub can be problematic, unless the condition is unequivocal, because the triggering disease may cause the victim to aspirate some liquid. Victims of homicide in a bathtub may present a clear injury pattern or – usually when the assailant is physically stronger and the crime is premeditated – may cause minor or no injuries with very subtle scene findings. In infants and children or those with a disability, bathtub drowning may raise issues concerning adequate supervision. Infanticide by drowning in a bathtub is sometimes followed by disposal of the victim in their crib or in the parental bed to stage a sudden infant natural death.

## Autopsy findings

The PM diagnosis of drowning can be a challenging task, especially when no reliable witnesses have provided any assistance regarding the events leading to death, and a full autopsy discloses no morphological changes associated with aspiration of liquid (see Table 27.2). One study conducted in Finland on 1590 bodies found in water showed that approximately 60 per cent of fatal drownings provide no definite circumstantial data or morphological changes that would allow a positive diagnosis of drowning [83,84]. In such cases, an autopsy diagnosis of drowning usually depends upon exclusion of any fatal traumatic lesions, medical conditions or type of intoxication. If appropriate, PM DNA analyses may exclude the presence of arrhythmia-related gene mutations. The diatom test, under certain conditions, can provide evidence supporting a diagnosis of drowning.

The main morphological changes associated with drowning are related to the penetration of liquid into the airways and consist of external foam, frothy liquid in the airways and lung overexpansion (Figure 27.6). Such changes are unspecific but may be valuable for the diagnosis of drowning. They must however, be evaluated within an appropriate investigative context. Conversely, a ‘negative autopsy’, especially in cases with advanced PM changes, cannot exclude the diagnosis of drowning, because time-dependent PM putrefaction efface or mask drowning-associated morphological changes.

The critical evaluation of morphological autopsy findings related to liquid penetration into the airways should account for three important, often overlooked, factors:

1. The various drowning scenarios that cause variable duration of the drowning process and of volume of liquid aspirated into the airways and lungs.
2. Unconsciousness due to medical conditions, injuries, intoxications, with persistent spontaneous ventilation leading to active aspiration of liquid.
3. Terminal gasping that may lead to agonal aspiration of liquid into the airways, in natural or injury deaths occurring in water (Table 27.3).

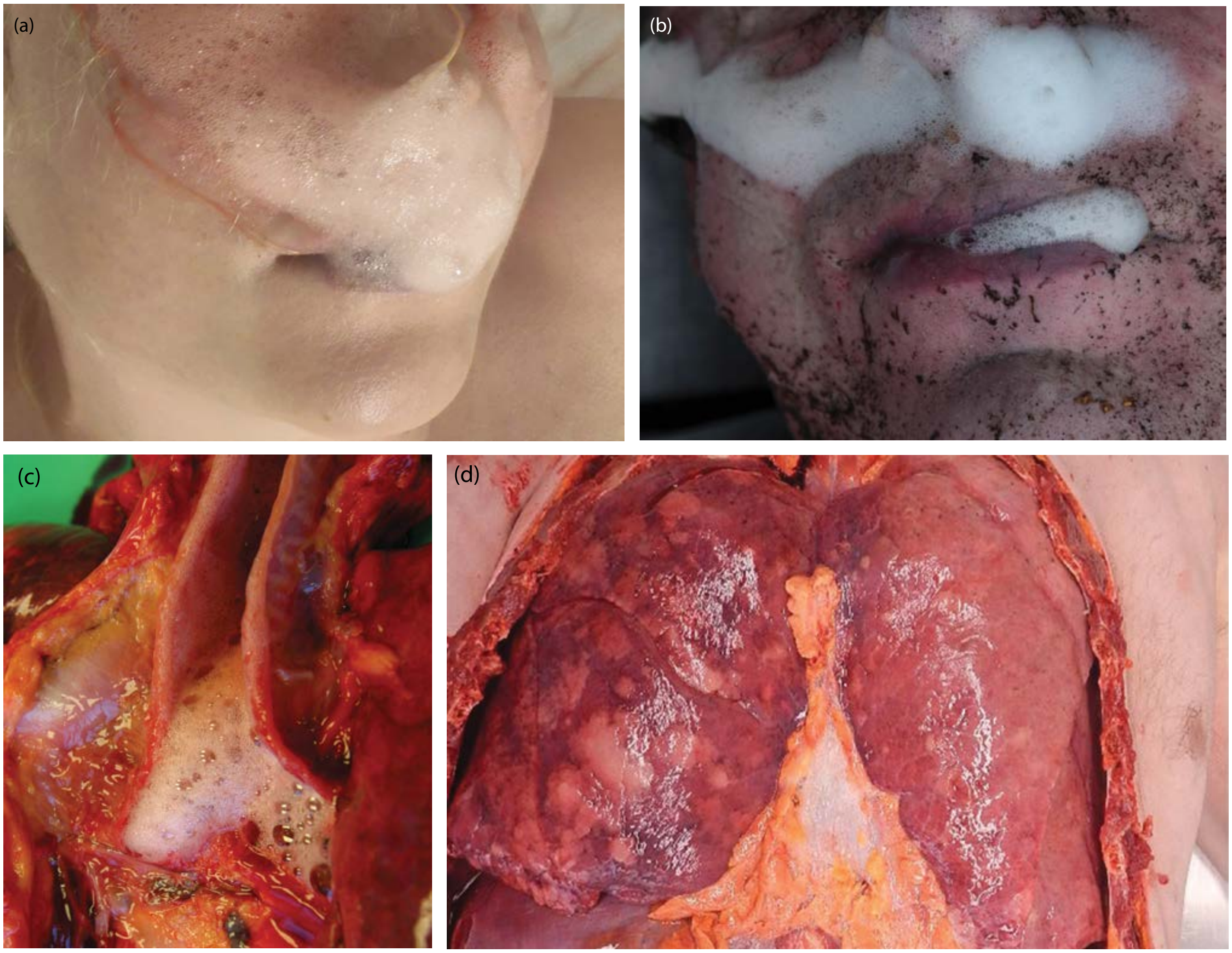


**Figure 27.6** Main macromorphological changes found in victims of drowning. (a),(b) External foam protruding from the nostrils and mouth; (c) frothy fluid in the airways; (d) overdistension of the lungs with overlap of their anterior margins. In a series of 20 131 consecutive bodies found in water, these findings were reported respectively in 18.5 per cent, 38.1 per cent and 38.9 per cent of the cases [81]. These changes may at times appear in deaths other than drowning.

**Table 27.3 Selected factors that may hamper or challenge the interpretation of drowning-associated autopsy findings**

| Factors | Mechanisms |
|---|---|
| Drowning scenarios<br>Submersion/immersion<br>Water temperature<br>Victim's age<br>Victim's medical condition | Duration of drowning process, volume of liquid aspirated |
| Terminal gasp<br>Fatal injury<br>Fatal disease | Agonal penetration of liquid into airways |
| Unconsciousness<br>Non-fatal medical conditions and injuries with spontaneous respiration | Penetration of liquid into airways |
| PM changes | Effacing morphological changes<br>Allowing penetration of liquid into organs |
| Prolonged submersion at depth | Mimicking lung changes<br>Allowing penetration of liquid into organs |

## Macroscopic changes

### External foam and frothy liquid in the airways

The penetration of drowning liquid into the respiratory system causes a reactive pulmonary oedema that, together with bronchial secretion and pulmonary surfactant, produces a white or blood-tinged frothy liquid. Respiratory efforts during the drowning process cause this frothy liquid to ascend toward the upper airways and eventually to extrude from mouth and nostrils, at times as sponge-like foam. In this frothy liquid, the fine air bubbles are initially resistant to collapse due to the presence of surfactant. Later on, fine bubbles coalesce into larger ones and, with the onset of putrefaction, frothy liquid can assume a red-brown discolouration, then fade and ultimately disappear with more advanced PM changes.

External foam and frothy liquid in airways are not consistently found in victims of drowning. Among 2031 consecutive drowning cases examined in southern Finland during 1987–2012, external foam was evident in only 18.5 per cent and frothy liquid in 38.1 per cent of cases [75] (Table 27.4). Similarly, in a recent study of 112 fresh-water drownings, external foam was detectable in 16 per cent of the victims [119].

**Table 27.4 Percentage of main PM macromorphological findings in bodies found in water with PM submersion time <24 hours ($n = 1111$), ≥1 day and <7 days ($n = 486$), and ≥7 day ($n = 434$), southern Finland 1987–2012**

| PM submersion time | External foam (%) | Frothy liquid in airways (%) | Overlap of lung margins (%) |
|---|---|---|---|
| <24 hours | 24.5 | 51.1 | 47.6 |
| 1–6 days | 19.1 | 37.2 | 43.2 |
| ≥7 days | 2.5 | 5.1 | 11.8 |

An absence of such external foam can be related to differing factors such as a low volume of drowning liquid aspirated and a limited production of pulmonary oedema or be due to external foam fading or disappearing owing to putrefactive PM changes. Moreover, external foam can be washed out during submersion of a corpse or be mechanically removed during the body-retrieval procedure, during any resuscitation attempts, and during transportation to the morgue. In bodies with no external foam at the nostrils and mouth, compression of the chest can eventually drive frothy liquid from the upper airways into the oral and nasal cavities. Transnasal endoscopy in victims of drowning provides, eventually, the possibility to examine and document a drowning victim's inner airways before autopsy.

External foam and frothy liquid in the airways are not specific for drowning, however, as it can be observable on occasion in cardiogenic pulmonary oedema, in drug intoxication, epilepsy and electric shock. One autopsy series ($n = 250$; fatal drug intoxication, myocardial infarct, acute asphyxia, gunshot wound) revealed that external foam appears in approximately 1 per cent and frothy fluid in the airways in 20 per cent of non-drowning deaths [83,84]. Although in these cases, the quantity is usually lower than in drownings, some non-drowning cases may exhibit external foam and frothy fluid in the airways that closely mimics that in drowning.

### Lung overinflation

The degree of lung overinflation and waterlogging in drowning is variable, and such changes can prove difficult to differentiate from those detectable in non-drowning deaths. Lung overinflation can also become pronounced to the point that it occupies entirely the pleural cavities, making their anterior margins overlap on the mediastinal midline under the sternum. The lungs are often crepitant, with their surface mottled and pale, with red and grey areas and sometimes visible alveolar distension. Once removed, the lungs often retain their shape without collapse due to intrabronchial frothy liquid, and the cut sections ooze foamy liquid spontaneously or after squeezing. Subpleural haemorrhages (Paltauf's spots) are unpredictable and not diagnostic of drowning.

Among the 2031 drowning victims examined in southern Finland (mentioned above), lung overinflation with the overlap of the anterior margins was a finding visible in 38.9 per cent of the cases (Table 27.4). In addition to PM findings, the lack of marked lung overinflation is likely linked to the pathophysiology of drowning. Pleural adhesions, too, can limit or prevent lung overdistension. Emphysematous bullae generally indicate chronic emphysema, as they do not usually newly develop during the drowning process.

Some authors differentiate lung morphology in fresh-water drowning (dry, light, emphysematous, anaemic) from their morphology in salt-water drowning (massively distended, swollen, heavy, waterlogged). However, this distinction seems a theoretical one, based on movements of hypotonic and hypertonic liquids across the alveolo-capillary barrier, rather than usefully reflecting actual observations at autopsy.

Lung weight in salt- and fresh-water drowning and in non-drowned controls overlaps, as well making lung weight of no practical utility for the diagnosis of drowning and for distinguish salt- from fresh-water drowning.

Common experience and a number of medicolegal studies suggest that, in approximately 10–15 per cent of drowning cases, the lungs appear normal and dry, with a low weight. Hence, during the 20th century, the term 'dry lungs' became increasingly popular among clinicians and forensic pathologists: they defined dry drowning as drowning without aspiration of liquid, aspiration being prevented by mechanisms such as prolonged laryngospasm and vago-vagal cardiac reflexes. Penetration of hypotonic liquid into the circulation in fresh-water drowning was also offered as a mechanism explaining dry lungs.

Some studies, using a cut-off total weight for both lungs of 1000 g, showed that the percentage of dry lung ranges between approximately 6 –8 per cent [69,101] and 10.4–16.8 per cent [23], with no significant differences between fresh- and salt-water drowning. However, a study focusing on 578 definite drownings with apparently dry lungs involving corpses with no putrefaction disclosed the presence in more than 98–99 per cent of the victims of other changes associated with liquid penetration (external foam, frothy fluid in the airways; diatoms in lungs and internal organs) [81].

Similarly, but in opposite circumstances, mechanisms other than liquid aspiration may give the lung a waterlogged aspect similar to that observed in drowning. For instance, mild pulmonary oedema has been described after swimming and diving, especially in cold water [50], and shock induced by a variety of natural causes can produce marked pulmonary stasis and oedema.

Finally, in natural or traumatic deaths occurring in water, terminal gasping may result in liquid entering the airways and complicating the distinction between true drowning and non-drowning deaths in water with agonal liquid aspiration. Lung overdistension mimicking that observed in drowning can also occur PM, when the body is for a length of time submersed at depth (0.2–0.3 atm) [117].

In conclusion, lung morphology varies across a spectrum that is not necessarily linked to the salinity of the drowning medium. Heavy, large oedematous lungs represent one end of the spectrum produced by liquid penetration into the lungs, with the other end represented by apparently normal, dry and low-weight lungs. This means that lung morphology at autopsy does not allow experts to achieve any definite conclusion as to the penetration of liquid into the airways in drowning. Differing body structure, pulmonary vital capacity, cardiac and respiratory functions, duration of the drowning process, and volume of liquid inhaled may each produce differences in lung morphology and weight.

Conflicting observations exist as regards the actual frequency of pleural effusion in drowning and the relation between pleural effusion, PM submersion time and lung weight [69,101,138,164]. Advanced putrefaction changes are generally not associated with marked pleural effusion.

## Swallowing and presence of liquid in the stomach

Water swallowing during drowning has long been a subject of investigation. Some authors maintain that drowning victims swallow a much greater quantity of water than they inhale [107], but autopsy data may prove inconclusive. At autopsy, the stomach of a drowning victim may be empty or show some watery liquid, liquid mixed with food, or food exclusively. When liquid swallowing occurs, the gastric contents poured into a receptacle separate into three phases (Wydler's sign): at the bottom, the solid components, above this, a clear watery liquid, and at the top, foamy liquid. Detection in the stomach of waterborne particles such as diatoms is inconclusive, because such particles can reach the stomach AM with food and beverages or can penetrate PM.

Investigation of stomach contents is of very limited value for the diagnosis of drowning, but in some circumstances, such as watery liquid in the stomach of a newborn, may raise the suspicion of drowning after birth.

Liquid swallowing causes increased risk of vomiting, spontaneously or during resuscitation, with aspiration of gastric contents leading to pulmonary infection and chemical irritation. Aspiration of vomit can hamper pulmonary resuscitation and contribute to electrolyte disorders. In drowning, penetration of liquid, vomiting and cardiopulmonary resuscitation may cause gastric mucosal tears. Data on the occurrence of emesis in fatal drowning are limited and to some extent controversial [11].

## Other changes

Liquid in the paranasal sinuses of drowning victims as well as haemorrhages in the temporal bones has been associated with drowning but are detectable also in deaths other than from drowning. If waterborne particles are detectable, liquid in the sinuses may indicate submersion but not necessarily drowning, because liquid may also penetrate PM the sinuses.

At autopsy, haemolytic staining of the aorta and the endocardium of the left heart has been reported in fresh-water drowning [146,166], but this change is difficult to assess objectively, is subject to PM modification and occurs also in non-drowning deaths.

Observation of a small, anaemic spleen with reduction in its weight has been a finding in drowning [52], but other studies have questioned its value [51]. Moreover, in large drowning series, spleen dimension and weight show an exceedingly wide range.

In drowning, muscular haemorrhages appear in the neck, upper extremities and chest, possibly as a result of muscular contraction during the drowning process or during agonal convulsion. Prospective studies show such haemorrhages in more than half their drowning victims [20,113]. A differential diagnosis between drowning-related haemorrhages and traumatic haemorrhages resulting from strangulation or other external violence is crucial. Drowning-related haemorrhages are generally subfascial and more elongated than those associated with external injuries [113], but this is not always the case.

Petechial haemorrhages in the periorbital region and in the conjunctiva are very seldom observable in drowning, but, surprisingly, such changes were reported in as high a proportion as 13 per cent of the cases in a series of paediatric drownings [133].

Vegetation, sand and other material manually grasped has been viewed by some as an indication that the deceased was alive when entering the water and eventually reaching the bottom. However, manually grasping foreign material may in principle occur during the formation of rigor mortis in corpses lying in shallow water or on the bottom.

## Forensic radiology

The diagnostic value of morphological findings detected by PM computed tomography (PMCT) has, at best, the same limitations as the changes detected at autopsy. PMCT can detect all major changes associated with drowning (frothy liquid in the airways, hypo- and hyperperfused lung areas, distension of the stomach due to swallowed water, liquid in sinuses and mastoid cells) [72,116,149,151]. In a virtual autopsy context, CT-guided coaxial cutting needle biopsy for obtaining internal organ samples for analysis of diatoms and other planktonic elements [123] is a noteworthy approach that deserves further testing.

## Injuries

Injuries detected in corpses found in water can be the unique cause of death, can contribute to drowning or can be unrelated to the death. Physical harm can occur before entering the water, at the moment of impact on the surface or while in the water.

Injuries sustained before entering the water occur in air, land and water-related traffic accidents or as a consequence of falling, jumping or being thrown from a dock, bridge or rock and striking a surface or fixed structure. Ligature, stabbing, cutting and shooting injuries in bodies found in water must be thoroughly considered to differentiate a homicide from a combined suicide.

Injuries due to the impact on the water surface are usually caused by falls from a great height, such as in a suicide by jumping from a bridge or in air- or land-traffic accidents. Such an impact can cause skin lesions, bone fractures and lacerations of internal organs. In shallow water, the impact on the bottom (such as in a swimming pool) may cause head, neck or spinal cord injuries.

While in water, the victim may sustain injuries by being washed by waves against rocks or a pier, by being struck by a boat or personal watercraft, or by being attacked by marine predators. Boat propellers cause skull fractures, dismemberment or parallel-cut wounds in the arms or other body parts [30,95,162]. In cold climates, a victim falling in water from an iced surface may sustain skin injuries on the face and hands during attempts to exit the water, grab the edges and again reach shore or just the surface.

During immersion, accidental, self-inflicted or homicidal electrocution may occur, in a bathtub or, less frequently, in an open body of water [21,31]. While some authors mention the incompatibility of certain injuries with water (burns, missile wounds, patterned injuries), the possibility exists that such injuries were sustained while the victim was partially immersed.

PM injuries found on a corpse retrieved from water include the following:

- Voluntary or accidental injuries produced before (dismemberment) or during cadaver disposal in water (skin abrasions while dragging the corpse ashore).
- Skin abrasions on the exposed surfaces (forehead, nose, back of the hands, knees, toes), as the result of current-induced drift and lift movements when the corpse lies in a semi-prone position in shallow water or at the bottom.
- Boat and personal watercraft injuries, especially by propellers.
- Other mechanical injuries (dashing against rocks, other surfaces or fixed objects).
- Aquatic-life depredation.
- Skull fractures in cold climates as a result of ice-induced bone stretching.
- Injuries during search and retrieval procedures.

Differentiation between AM and PM injuries can be challenging. During the early PM period, blood in a vital wound may be washed away from subcutaneous tissues, giving the wound a PM appearance. In addition, some wounds may occur perimortally before submersion. Moreover, injuries such as ligature marks or conjunctival petechiae may fade or vanish in water even before the onset of putrefactive changes, and late decomposition can efface AM wounds or modify their characteristics.

## Microscopic changes

Histological changes in the lungs associated with drowning consist of acute emphysema, especially in the subpleural regions, with alternation of areas with overdistended alveoli, thinning or lacerations of alveolar septa, capillary congestion, intra-alveolar oedema and haemorrhages, with areas of atelectasia. Diagnostic limitations mentioned above related to macroscopic lung changes also limit the practical utility of microscopy for diagnosis of drowning. Analytical morphometry has been tested to diagnose acute emphysema in drowning, but in only a few experimental studies [40,68]. The uneven distribution of drowning-related changes represents a further limitation complicating the interpretation of changes in routine cases, with lung findings in those drowned that may well overlap findings in non-drowning victims. At times, exogenous waterborne particles appear in the bronchioles and alveolar spaces, but discrimination between AM and PM penetration is challenging, especially when microscopy is limited to a few specimens from each lobe.

Alveolar macrophages, pulmonary surfactant, and, more recently, lung aquaporins, a family of water-channel proteins, have been studied mainly in experimental models of drowning by means of histology and molecular biological methods [9,55,64,70,167].

Ultrastructural, mostly experimental, studies performed by transmission electron microscopy (TEM) and scanning electron microscopy (SEM) have disclosed changes specific for salt- and fresh-water drowning [12,17,104,111,118,127,145]. During the 1990s, a few ultrastructural studies were focused on tracheal cilia [114] and penetration of tracers [6] and diatoms [82] through the alveolo-capillary barrier.

## Laboratory tests

Laboratory testing for drowning has been investigated for over a century to study the pathophysiology of drowning and to find reliable criteria for its PM diagnosis [80]. Extensive reviews of laboratory methods for the diagnosis of drowning have been published by Moritz [102], Reh [117] and Lunetta and Modell [80]. During the last decades, laboratory methods have been critically appraised, with some investigators reiterating reports as to their potential and others disputing their validity. However, as most studies have flawed designs, yield divergent results and are limited to experimental conditions or to small series, laboratory tests are nowadays considered as having no or scarce practical value for diagnosis of drowning. Yet, in individual cases, laboratory tests aimed at the identification of exogenous marker tracers of the drowning media may be useful to point to a homicidal drowning followed by disposal of the body (e.g. the finding of bath salts in newborns or babies drowned in a bathtub and transferred to cribs).

Laboratory tests can be divided into those that investigate:

1. *Changes in blood properties and composition:* In this group, cardiac biventricular $Cl^-$ concentration has been repeatedly investigated, based on the hypothesis that high $Cl^-$ concentration in the left ventricle compared with the right ventricle occurs in salt-water drowning, with the opposite levels in fresh-water drowning [47,102,129].
2. *Penetration into the circulation of exogenous substances:* Numerous solutes, including electrolytes, inorganic debris, bath salts, pollutants, zoo- and phytoplanktonic elements and microorganisms, may enter the airways and circulation following aspiration of the drowning liquid. Among ionic tracers, the most studied are strontium ions ($Sr^{2+}$) due to the high seawater to serum $Sr^{2+}$ concentration ratio [4,5,110]. Marine $Sr^{2+}$ concentration variability in differing geographical areas (e.g. higher in the Mediterranean than in the Baltic Sea) and potential passive PM diffusion of $Sr^{2+}$ can both limit this method's practical applicability.
3. *Penetration into the circulation of artificial tracers:* Particulate matter, chromatic substances, radio-opaque media and isotopes have been used to investigate in experimental models the penetration of the drowning media into the respiratory tract, the circulatory system and peripheral organs [80], but these methods obviously have no diagnostic utility.

Some laboratory testing has focused, also recently, on matrices other than blood, such as vitreous humour [45], pericardial and pleural liquid [93], sphenoid sinus liquid [54] and even teeth [41].

In experimental drowning and routine caseworks, aquatic microorganisms, including bacteria and algae, have been identified in matrices such as blood, lung swabs and internal organs. Microorganism detection by incubation in selective media or by polymerase chain reaction (PCR) techniques may assist in the diagnosis of pneumonia; results can be compared with those of the alleged drowning media [136]. In addition to DNA fragments specific for aquatic bacteria, the 16S ribosomal DNA gene (16S rDNA) has served as a target for bacterial and planktonic elements [56,61,62,66,140]. PCR probes for have also been tested in routine caseworks [115,122,123].

## Diatom test

The diatom test is based on the assumption that, during the drowning process, diatoms reach the lung during aspiration of liquid, and, if valid cardiocirculatory activity exists, distribute themselves through the bloodstream into closed organs [82]. Conversely, in a corpse only entering the water after death, diatoms will not reach closed organs.

Diatoms are waterborne eukaryotic unicellular or colonial algae in size from 2 μm to over 500 μm (Figure 27.7). The great majority of diatoms penetrating the alveolo-capillary barrier and reaching closed organs are of size <60 μm, although some investigators report diatoms up to 100–160 μm [74].

The siliceous diatom's cell wall is composed of two interconnected units (valves), one structure on which the taxonomic classification is based (more than 30,000 species) [92,121]. The valve's pattern either of radial symmetry or of

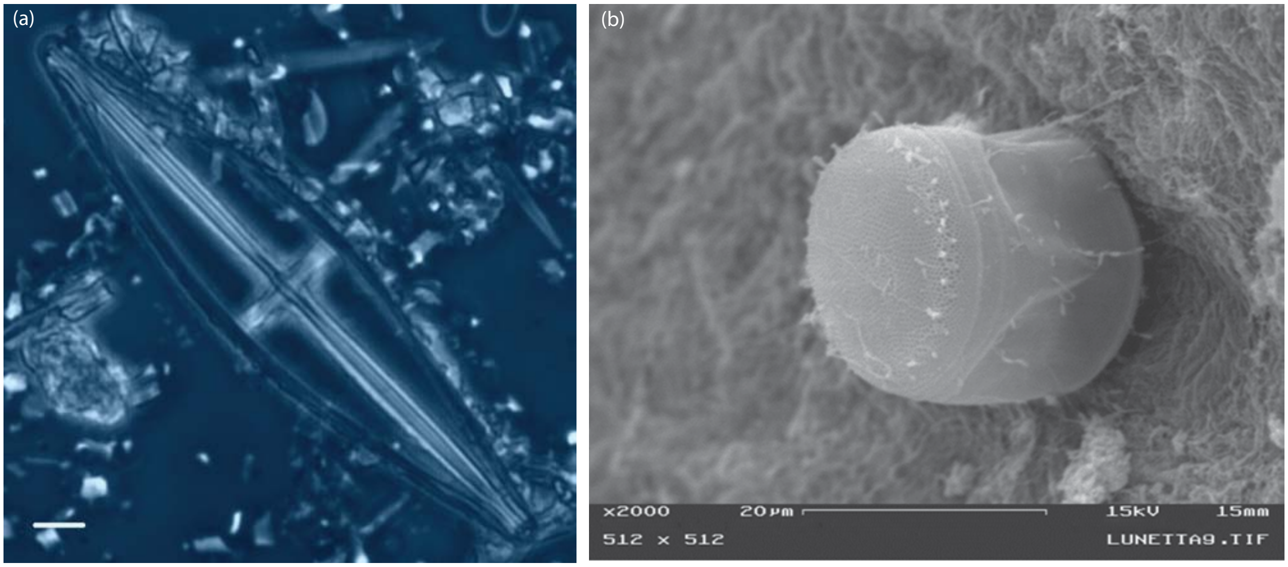


**Figure 27.7** (a) A fresh-water diatom, *Stauroneis phoenicenteron,* recovered from a lung specimen after acid digestion (scale bar 10 μm; phase contrast). In drowning cases, the number of diatoms recovered after digestion from a victim's 10–15 g lung tissue may vary from less than 100 to more than 1000. Factors affecting the number of diatoms in the lungs of drowning victims include the original concentration of diatoms in the drowning medium and the volume of aspirated liquid. (b) A diatom, *Thalassiosiria baltica,* lying on the pleural surface *(*SEM ×2000*);* experimental conditions: the cadaver's lungs were injected prior to autopsy via tracheostomy with a solution containing a bicellulate diatom culture (*Thalassiosira baltica, Thalassiosira levanderi*).

elongation provides the basic distinction between centric and pinnate diatoms. Quantitative analysis and taxonomic comparison between diatoms in putative drowning media and in the victims' organs can assist in the diagnosis of drowning, in discrimination between fresh- and salt-water drowning and in determination of the drowning site.

In diatom analysis, several procedures of sample preparation are currently available. In routine cases, these include organ sampling (brain, lungs, liver, kidney, bone marrow) at autopsy, tissue destruction (nitric or sulfuric acid, proteinase K, Soluene®350), centrifugation for diatom concentration, and specimen analysis by light microscopy [74].

Contradictory opinions exist on the diagnostic value of the diatom test for drowning. The main criticism stems from the discovery of diatoms in non-drowned corpses. In principle, due to the ubiquity of diatoms in water, air and soil, false-positives can result from AM penetration of diatoms (ingestion of diatom-laden beverages or food, inhalation of aerophilic diatoms, swallowing or aspiration of water by swimmers or divers) and PM penetration during submersion (through AM wounds and PM artefacts; at high hydrostatic pressures). Contamination may also occur during tissue sampling at autopsy and in the laboratory during procedures for sample preparation; these include instruments, gloves, paper, water supplies, reagents and glassware, which are all potential contamination sources.

Quantitative data on false-positive cases remain conflicting, however. Most investigations report few or no diatoms in the peripheral organs of those non-drowned, but one Danish study, repeatedly cited in the medicolegal literature but only based on four non-drowned corpses, reports up to 194 valves/cm$^3$ lung [39]. Two more-recent studies, performed with standardized protocols to avoid contamination, showed in non-drowned corpses the absence of diatoms or their merely sporadic presence (<1 diatom/body) [16,79].

The diagnostic value of diatoms in the lung alone is also debatable. Some authors state that finding diatoms in the lungs but not in closed organs suggests that (the possibility of) passive penetration cannot be excluded; other investigators suggest that a sufficient number of diatoms, defined as >20 diatoms/slide, is diagnostic for liquid aspiration. Similarly, no consensus exists on the number of diatoms reaching closed organs in drowning: some researchers speak in terms of dozens and others of single diatoms. Diagnostic cut-offs have been set by some authors at 5 diatoms/slide per 100 μl pellet for internal organs [73], whereas others have proposed separation values of up to 20–40 diatoms/5 g in bone marrow [60].

Comparisons of results among studies on the diatom test for drowning are hindered by flawed study designs, difficulties in transposing findings from animal experiments to human corpses and lack of standardized protocols. Shortage of information on any diatom content of the drowning media, extraction and centrifugation procedures, and aliquots of tissues sampled and mounted on the slide are additional limitations. Systematic studies on false-positive diatom tests in human bodies submersed after death are lacking, although the main application of the test is distinguishing victims who entered the water alive from victims whose bodies were disposed in water after death.

On the other hand, absence of diatoms in corpses recovered from water does not exclude drowning as the cause of death. False-negative cases may be related to a low diatom concentration in the drowning media, to a low volume of liquid aspirated, to an insufficient aliquot of tissue sampled, or to the loss of diatoms during sample preparation

When conventional diatom testing yields insufficient evidence of drowning, PM investigations can be extended to other waterborne planktonic elements [26,88]. During the past 20 years, molecular studies by means of 16S rDNA and DNA probes have been tested in drowning for detecting bacterio- and other planktonic elements (see 'Laboratory tests'). The higher sensitivity of the DNA methods compared with the classical diatom method has been emphasized. However, DNA methods do not allow the by-passing of false-positivity issues, and, in principle, these methods can more easily yield contamination, especially when a corpse lies in water for a long time.

In conclusion, giving an expert opinion on the results and validity of the diatom test in a putative drowning case requires a judicious approach. Obviously, the mere finding of a few diatoms in human bodies does not endorse a diagnosis of death by drowning. Any acceptable results which can counter the criticisms levelled against the test require sample analysis by an expert diatomologist, taxonomical concordance between diatom content in the body and putative drowning media, and strict protocols to avoid contamination during sample preparation [86]. Standardized studies with a large number of cases that can eventually confirm well-defined separation values for diatoms between actual drownings and non-drowned corpses, either found on dry land or disposed of in water after death, may further validate the results of a diatom test.

## Contributing factors

The most common individual contributing factor of drowning is alcohol. Several studies conducted in HICs have disclosed the association between alcohol and fatal unintentional drowning in up to 25–50 per cent of the cases. In Finland, as many as 60 per cent of victims of fatal unintentional drowning are ethanol alcohol-positive and more than 50 per cent have a blood alcohol concentration (BAC) over 100 mg/dl. [108]. Alcohol contributes to drowning by altering psychomotor performance and cognitive processes, possibly causing a person to fall into water, disregard safety procedures in aquatic settings and/or operate a boat improperly. Once in water, the victim's alcohol-intake can hamper their ability to swim and reduce breath-hold time.

Recently, two studies conducted in northern Europe have highlighted the potential role of medicinal drugs or drugs of abuse as a factor contributing to unintentional drowning. Many psychotropic drugs have effects on cognition and psychomotor functions similar to those of alcohol. A Swedish study [2] demonstrated that 22 per cent of victims had in their blood at the time of death one or more psychotropic drugs, mostly benzodiazepine and antidepressants. A second, more in-depth study [108] conducted in Finland, disclosed that psychotropic drugs may play, alone or in association with alcohol, a significant role in up to 14.5 per cent of accidental drownings. In deaths by drowning, some authors have emphasized the potential of drug-induced long QT-syndrome [153,154].

Pre-existing medical conditions may be the sole cause of death in water or may contribute to drowning, for instance by causing the victim to fall into water or during swimming under the effect of physical exertion or cold. Few studies examine the actual risk posed by natural diseases in causing sudden death or contributing to deaths in aquatic settings, with the exception of epilepsy. Recently, a cross-sectional population study demonstrated that, with the only exception being epilepsy, pre-existing medical conditions do not increase the risk of drowning in children [42]. In victims with severe, but not acute, medical conditions, it may be impossible to disentangle their effects from those of liquid aspiration, as liquid may penetrate the airways also in an unconscious victim with spontaneous respiration or during terminal gasping.

Genetically determined cardiac channellopathies, especially long-QT syndrome, have been associated with drowning in healthy individuals who are good swimmers, but the actual frequency of gene mutations causing cardiac arrhythmias among drowning victims is unknown. Differently designed studies have yielded contradictory results [78,85,139,147,148].

## Manner of death

The vast majority of drowning is unintentional, but drowning can also be the result of a suicide or a homicide and, in some cases, the manner of death cannot be ascertained even after full police and medicolegal investigations.

### Accidental

The circumstances and demographic profiles of victims of accidental drowning vary across continents, countries and even within regions of the same country. The diagnosis of accidental drowning is often reached by means of witness accounts and by exclusion of other manners of death through police investigations and full autopsy. However, especially in LMIC, a diagnosis of accidental drowning is often established solely on the basis of a corpse being found in water and lacking significant physical injuries at external examination.

## Suicide

Suicide by drowning is less common than unintentional drowning. Its incidence may be underreported in countries with no thorough police and medicolegal investigations or with a significant percentage of suicides classified as undetermined deaths. Epidemiological data on suicides by drowning are available for many countries by means of the external ICD-code (X71) via the WHO Mortality Database and national statistical offices. Several medicolegal studies have examined circumstantial and individual characteristics of suicides by drowning in North America, Europe, Asia and Australia.

Suicide notes, self-inflicted injuries such as superficial hesitation marks on the wrist or more severe injuries related to a combined suicide (strangulation, deep cut and incision wounds, gunshot wound, electrocution) and to witnessing are among the factors usually leading to a diagnosis of suicide. The percentage of witnessed cases (approximately 10%) is, however, much lower than in unintentional drowning. The percentage of suicidal drowning with farewell notes ranges in different series from approximately 14 per cent to 37 per cent. A verbal equivalent to a suicide note just prior to death has been reported in up to 25 per cent of suicidal drownings [95]. Other factors may suggest suicide, such as previous suicide attempts, suicide ideation and psychiatric morbidity, but these must be appraised judiciously; such characteristics can be evident or present also in victims of accidental or homicidal drowning [85].

Consistent with suicidal drowning may be the following: attachment of a heavy weight such as an anchor to a body, self-weighting by filling pockets with stones or by carrying bags containing heavy objects, binding one's hands or feet with ligatures or other restraining devices. In these cases, investigation of the origin of the weight and experimentation involving whether the victim could have applied the weight or the ropes unassisted is necessary to differentiate these cases from homicide by drowning or body disposal in water [34,144]. A suicide pact including drowning or homicide–suicide with drowning has been reported [7,94].

## Homicide

Homicidal drowning is often perpetrated by a physically stronger assailant against a weaker victim, generally a child, or an adult incapacitated by alcohol, drugs or disease. The setting is frequently a bathtub or a body of water with shallow water. Homicide by drowning can, however, result from simply pushing or throwing—unobserved—a person taken by surprise into deep water. In these cases, the victim's possible lack of swimming skills and the setting may offer the victim no chance for self-rescue. The scene investigation, especially in premeditated homicide, will often lack any clue, and the victim will show no or only mild injuries. However, bruises on the shoulders or back should be sought also by dissecting subcutaneous tissues, as these may suggest pressure by the assailant's fingers and hands to keep a victim underwater, especially if they were struggling. Manslaughter homicide may occur during play or as a joke, if no effective attempt is made to rescue the victim. Conversely, when homicidal drowning is the final stage of an assault by other means (beating, strangulation, stabbing), physical injuries are evident, and a crucial issue is the assessment of whether the victim was still alive when they were sinking below the water surface.

Drowning as a form of fatal child abuse can be difficult to distinguish from unintentional drowning, or, when the corpse is moved to a crib or bed, from a natural death. Similarly, drowning may occur in a newborn when delivery take place under adverse conditions, for instance in a bathtub, with the corpse being moved after death to a dry location. In these cases, it is crucial to determine whether the victim was alive or dead at the time of delivery and, in the former case, whether the cause of death was drowning. Drowning has been described also in the context of sexually oriented breath-holding rituals with controlled submersion. If the ritual was assisted by bystander(s), the case should be investigated as a potential homicide [77,89].

### Disposal of corpses in water

The differential diagnosis between drowning and body disposal in water after a homicide perpetrated on dry land may pose specific challenges in corpses with advanced PM changes and when no clear injuries are detectable.

Disposal of corpses in a water setting aims essentially to conceal the body with the expectation it will remain under water or will be transported far away. However, such attempts frequently fail—even if the victim carries considerable extra weight, development of putrefactive gases may then create enough buoyancy to cause the body to ascend to the surface. Staging a homicide by drowning as a natural, suicidal or accidental death in water may also involve the disposal of a corpse from an aquatic setting to another (e.g. from a bathtub to an outdoor natural body of water). Moreover, a prolonged interval between a homicide and body retrieval from water may greatly hamper victim identification, evaluation of time of death and assessment of cause of death.

The disposal of a victim into water after a crime perpetrated near a body of water does not require complex action, at times only dragging the corpse from the shore to the water or dropping it into water. Conversely, when the murder is perpetrated far away from the site of concealment, disposal requires more elaborate actions such as the body being carried in a vehicle, weighted, placed in a sack and even dismembered.

## Undetermined

The manner of death in drowning can be placed in the category 'undetermined' when it is unclear whether it has been unintentional or purposely inflicted. Inherent difficulties in interpreting the circumstances of drowning or linking them to the victim's individual profile may hamper diagnosis of the manner of death, some investigators being in these cases more prone than others to classify drowning as 'undetermined'. Undetermined drowning rates vary also across countries, based on levels of evidence required by judicial systems to assess the intent, by inadequate transfer of data to national statistics offices, and low frequency of medicolegal investigations. In countries applying the criminal standard of proof 'beyond any reasonable doubt', a number of actual suicides by drowning may be classified as 'undetermined'.

## Conclusions

Mortality and morbidity in aquatic settings represent a significant public health and medicolegal issue, the investigation of which necessitates a multidisciplinary approach including expertise in swimming instruction, aquatic safety, behavioural sciences, pathophysiology, medicine, forensic science and pathology. Such an elaborate approach clearly has a much higher potential than does exclusive police and medicolegal investigation.

Many authors reiterate that the diagnosis of drowning is one of exclusion. While this is often correct, thorough PM investigation may at times corroborate a diagnosis of drowning and substantiate the role of contributing factors leading to death in water. However, small series and costly but poorly designed studies based on a constantly growing array of new markers that can hardly be used routinely in forensic casework have limited tangible value in court and do not improve our capacity to ascertain the cause of death in bodies found in water.

Court assessment of drowning incidents should evolve from personal views to evidence-based concepts. From a medicolegal standpoint, achieving this objective requires well-designed studies aiming to test in large drowning series the specificity and sensitivity of morphological changes and selected laboratory markers compared with those of non-drowned controls, either deceased on dry land or dumped into water.

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# 28 Autoerotic Asphyxiation

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## Introduction

Defined by Byard and Bramwell in 1991, autoerotic asphyxia denotes death resulting from failure of a release mechanism of the device, apparatus or prop designed to attain cerebral hypoxia for heightened arousal [15,31,61,66,68]. Forensic specialists often misdiagnose these deaths as suicides; however, evidence at the death scene often reveals a nude victim surrounded by pornographic material who had no intention of ending their life [13].

The estimated annual incidence of sexual asphyxia is 2–4 cases per million in the United States [11], 1–2 cases per million in Scandinavia [34], 0.2–0.5 cases per million in Canada [54], 0.3 cases per million in Australia [18], and 0.14 cases per million in Sweden [18]. The most common mechanism of autoerotic death is by ligature asphyxiation [54,61,66,68]. Experimentation with other methods such as plastic bag asphyxia [39,40,51,63,68], inhalation of noxious chemicals [27,35,36,44,46,47,68,69], aqua-eroticum (autoerotic drowning) [32,60,71] and electricity [2,19,42,65,68,70] have resulted in a fatality while striving for sexual gratification.

Hazelwood and colleagues [29] have determined the following five criteria for an autoerotic death: a method of attaining sexual arousal with a (1) well-defined self-rescue mechanism, (2) a solitary activity which uses (3) sexual fantasy aids, (4) previous autoerotic behaviour, and (5) no suicidal ideation. Ligature hanging involves a decreased circulation of the cerebral arterial blood and venous return leading to hypoxia and hypercapnia [52]. This distorted consciousness heightens sexual arousal. Four mechanisms interact to contribute to an autoerotic fatality: neck compression, airway obstruction, chest compression and oxygen deprivation [31].

Individuals who participate in autoerotic behaviour strive to attain the perfect combination of these four features to heighten sexual arousal. An individual with impaired consciousness or someone with minimal autoerotic experience may unintentionally suffer immediate syncope or coma by carotid sinus pressure [52]. Death may result from failure of either the physiological mechanism or the self-rescue device, or the distortion of the participant's judgement [29].

Asphyxia may be divided into four distinct categories [72]:

1. Compression of the neck, as seen in hangings and strangulations.
2. Obstruction of the airway associated with smothering by a plastic bag.
3. Mechanical compression of the chest.
4. Exclusion of oxygen by inhalation of another gas.

Autoerotic asphyxia with its myriad methods to heighten sexual arousal incorporates these four mechanisms. Central nervous system damage confirmed microscopically occurs 30–40 seconds after oxygen deprivation [72]. Cardiac function may continue for 10–20 minutes after respiratory arrest.

The 'erotized repetitive hanging' syndrome coined by Resnik in 1972 [52] is marked by a young adult Caucasian male who engages in a solitary masturbatory act utilizing bondage that compresses the neck. These individuals may be nude or wear women's clothing, and pornography in the form of magazines or videos may be discovered at the death scene. There may be evidence of ropes, chains or leather binding the body and/or the extremities and/or the genitals. As this behaviour is clandestine, the participant often uses padding around the neck to hide any injuries inflicted by the ligature. Families of the victims of autoerotic asphyxia deny any suicidal ideation prior to death.

This chapter delves into the secretive behaviour of autoerotic asphyxia. We initially discuss the differences between 'typical' and 'atypical' autoerotic death and highlight the various methods of achieving sexual gratification. We then present the profile of the participants of autoerotic asphyxia, including the age, race, gender, marital status, and location of discovery of the body. Special attention is focused on the variations between male and female victims with respect to attire and pornographic materials found at the scene.

The next section of the chapter deals with the release mechanism designed to extricate an individual from an autoerotic situation prior to death. Participants in autoerotic asphyxia often visualize a fantasy scenario to fulfil their erotic desires. We describe the various items that may be utilized including cross-dressing, pornography and bondage apparatus. The next section classifies autoerotic asphyxia as a paraphilia. We subsequently discuss the importance of determining the manner of death in equivocal cases of autoerotic death and then examine the non-specific features that may be observed during postmortem examination and toxicological analysis in cases of autoerotic death. We conclude by analyzing the early agonal sequences discerned from non-lethal filmed ligature strangulations and hangings of an autoerotic asphyxia participant.

**Table 28.1 Characteristics of 'typical' autoerotic asphyxia**

| |
|---|
| Caucasian and unmarried male |
| Heterosexual |
| No evidence of psychiatric disturbance |
| Young adult |
| Social loner and an introvert |
| Excels in work and at school |
| Unwitnessed |
| Body discovered in an isolated or secure environment |
| Sexual fantasy aids and props, including cross-dressing, pornographic materials, mirrors, bondage and sexual paraphernalia |
| Asphyxiation by neck ligature with or without visible furrow |
| Protective padding around neck to prevent bruising or abrasions |
| Positional or mechanical release mechanism |
| Evidence of previous experience |
| Non-contributory postmortem toxicological results |
| No suicidal intent |
| Accidental manner of death |

## 'Typical' versus 'atypical' autoerotic death

'Typical' autoerotic death refers to ligature asphyxiation which comprises the majority of these cases [54,66,68]. Table 28.1 characterizes the 'typical' autoerotic asphyxia participant [31,67,75]. 'Atypical' autoerotic death encompasses the remainder of the asphyxiation mechanisms such as plastic bag asphyxia [39,40,51,63,68], inhalation of noxious chemicals such as propane, butane and nitrous oxide [27,35,36,44,46,47,68,69], aqua-eroticum [32,60,71] and includes other acts designed to attain sexual fulfilment, such as electricity [2,19,42,65,68,70], injection of air into the penis leading to generalized subcutaneous emphysema [74], insertion of a crochet needle into the urethra in a woman [22] and metal devices or a plastic bottle constricting and/or inserted into the penis [1,5,23]. In certain circumstances, individuals have selected more than one autoerotic method to attain sexual arousal. One 36-year-old male's autoerotic death was caused by neck strangulation, suffocation by a plastic bag and vagal stimulation due to a foreign body in the rectum [3].

## Profile of participants

The majority of victims of autoerotic asphyxia are Caucasian males younger than 40 years of age [7,8,10,18,31,37,43,61,66,76] (Table 28.1). Children as young as 9 or 10 years old have succumbed to this behaviour [7,8,61]. Adolescents who play asphyxial or choking games may progress to participating in potentially fatal autoerotic behaviour, suggesting an awareness and intervention by paediatricians and emergency physicians [21,24]. At the opposite end of the age spectrum, a nude 87-year-old man encircled by a bondage of ropes was discovered hanging by a belt in his living room [59].

Sauvageau and Racette [61] performed an extensive review of autoerotic deaths in the literature from 1954 to 2004. Of the 57 studies fulfilling the criteria of an autoerotic death, there was a total of 408 cases [61]. The vast majority of the victims succumbed to asphyxia by hanging, ligature, plastic bags, chemical substances or a combination of these methods. The remaining 10.3 per cent of cases were deemed to be 'atypical' autoerotic fatalities and consisted of electrocution (3.7%), overdressing/body wrapping (1.5%), foreign body insertion (1.2%), atypical asphyxia method (chest compression, inverted suspension/abdominal ligature, immersion/drowning and smothering) (2.9%) and miscellaneous (1.0%). The latter category comprised four cases: a man who masturbated with a vacuum cleaner and died of a heart attack due to atherosclerotic disease [33]; a man who used the heat from a table lamp between his thighs to increase his sexual enjoyment, and died from electrocution [73]; a man engaged in coprophilia who was found nude covered in faeces with pornography nearby and whose death was attributed to ischaemic heart disease with myocardial fibrosis [68]; and autoerotic Russian roulette whereby a man was found with a gunshot wound to his head with evidence of masturbation prior to the gunshot [68].

The wide variation in classification of asphyxial deaths results in lack of uniformity of data [58]. Sauvageau [53] revisited the common methods of autoerotic death based on the standardized classification of asphyxia. Ligature strangulation is classified as hanging when the pressure on the neck is applied by a constricting band tightened by the gravitation weight of the body or part of the body [58]. In this respect, only two cases of autoerotic ligature strangulation in their review study from 1954 to 2004 should continue to be classified as such, with the remainder newly classified as hangings. Sauvageau proposed that hangings be included as a typical autoerotic method, while ligature strangulation should be an atypical autoerotic practice [53].

Several striking differences are observed between male and female victims of autoerotic asphyxia when investigating the death scene [14,16,17]. Men are more likely to cross-dress, employ elaborate devices and props designed to produce real or simulated pain and utilize autoerotic materials [16]. Contrarily, women are usually discovered nude with a single ligature that has been tightened by lowering the body or by pulling on the attached cord tied to the hands or legs [16]. In addition, autoerotic paraphernalia is often absent at the scene in female cases. Due to the paucity of distinct autoerotic characteristics at the scene of female autoerotic deaths, investigators may initially presume a homicidal or suicidal manner of death [17,62,68]. While Behrendt et al. [6] concur that female autoerotic deaths are rare, they noted a close similarity between the scenes of both male and female autoerotic fatalities. In addition, Behrendt and colleagues attributed the low number of women participating in autoerotic asphyxia to either their more cautious nature or incorrectly diagnosing this cause of death at the scene or at autopsy.

Most victims of autoerotic asphyxia are discovered indoors by family or friends on their personal property [10,61,66]; however, others have been found in the victim's car, a hotel room or a public parking lot [10]. In our study of 16 victims of autoerotic asphyxia, most (75%) of the subjects were discovered hanging in their home, while the remainder solely used bodily movement to attain hypoxia and sexual satisfaction without suspension [66]. The majority of participants of autoerotic asphyxia are single [7,8,31,66,76]. Evidence discovered at the scene may suggest repetitive autoerotic behaviour, such as worn ropes or indentation of rafters at one's residence [16,66].

## Release mechanism

An analysis of the ligature encircling the victim's neck offers a valuable clue as to the release mechanism selected to avoid a fatality. There is often a 'failsafe' mechanism whereby the participant may quickly release the tension on the binding and thereby lessen the hypoxia [12]. The 'self-rescue' mechanism may denote either the participant's own judgement of the appropriate timing to release the constricting pressure that is inducing hypoxia (by manipulating their body position in a specific position) or a complex release mechanism consisting solely of ligatures irrespective of the body position [67]. These two mechanisms may be referred to as positional versus mechanical in nature. This 'self-rescue' mechanism may prove unreliable and fatal in situations when a chair on which the victim stands falls over or if consciousness is lost, leading to increased ligature pressure from the uncontrolled weight of the body [12].

A common release mechanism is the slipknot [66]. The ligature may extend from the neck to encircle the victim's wrist or the decedent may have grasped the ligature in their hand. A participant may desire to hide any rope burns or bruises about the neck to conceal their surreptitious behaviour and may select protective padding between the ligature and neck, such as a towel, pillow, cloth wrap and Ace™ bandage (Figure 28.1) [12,16,66].

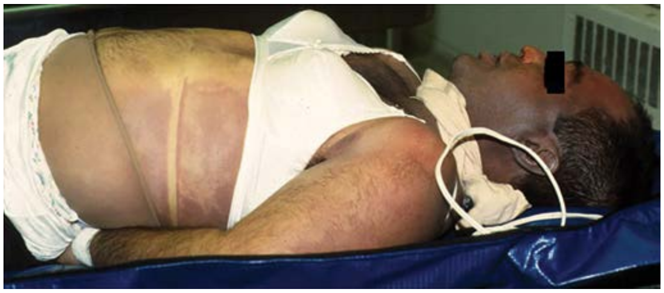


**Figure 28.1** Protective padding hides the ligature marks on the neck. (With kind permission from Wolters Kluwer Health, Inc.: Shields et al. [66].)

## Fantasy scenario

Participants of autoerotic asphyxia strive to heighten their sexual arousal by creating a fantasy scenario. A plethora of erotic items may be selected including cross-dressing, viewing pornography and bondage devices (Figure 28.2) [66]. Men may don high-heeled shoes, cloth-stuffed brassieres and black lace panties (Figure 28.2) [66]. To complete the fantasy scenario, individuals may desire pornographic magazines, books and videos (Figure 28.3). Apparatus used for bondage of the extremities ranges from duct tape, belts, leather straps, ropes, chains, metal bars, locks and rubber items to brass chains (Figure 28.4) [10,37,66]. In addition, men may be discovered with intricate penile binding materials (Figure 28.4). We reported a case of a 40-year-old man whose lower extremities were bound with duct tape as well as a rope encircling the legs and extending to encircle the neck (Figure 28.5) [66]. Duct tape surrounded the tip of the penis and scrotal sac bilaterally and these, in combination with the lower extremities, were shaved closely (Figure 28.5). He wore a tank top, white bra corset, black stretch female skirt and white high-heeled women's shoes. There was a silver-coloured ball in a pierced left ear, cosmetics on the left side of the face, a left bra cup filled with red cloth material, and bright pink fingernail polish on his left fingernails – all of which may have reflected his dichotomous lifestyle. His surreptitious autoerotic

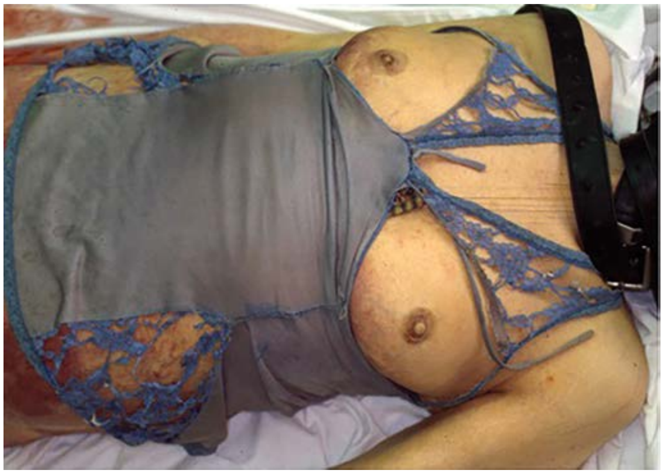


**Figure 28.2** A 44-year-old with exogenous hormonally created gynaecomastia was discovered wearing lace lingerie and a pair of black lace panties. (With kind permission from Wolters Kluwer Health, Inc.: Shields et al. [66].)

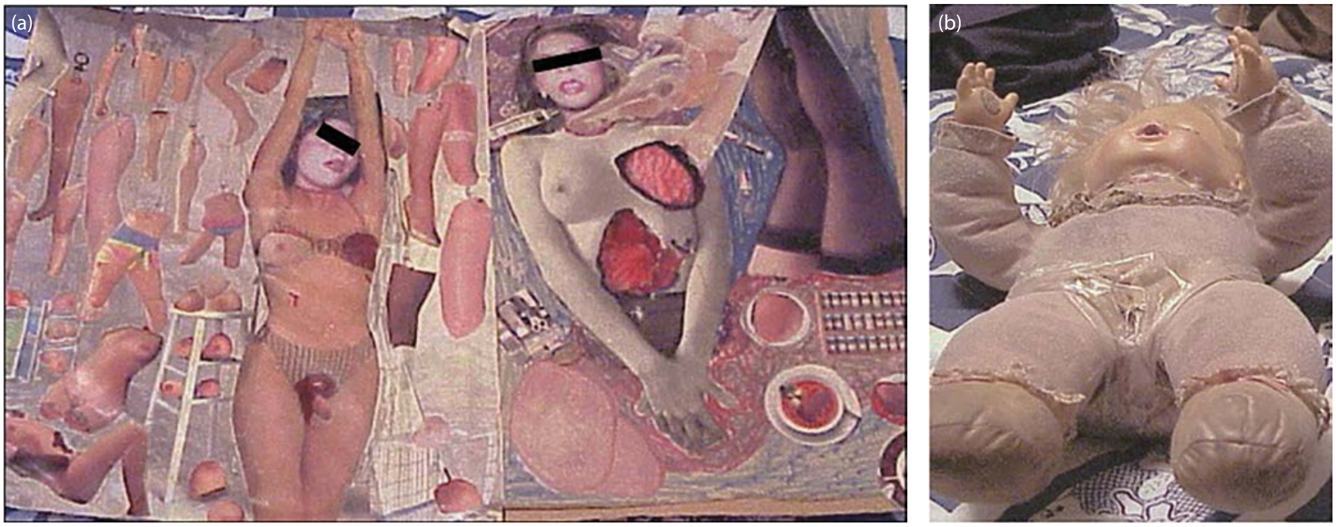


**Figure 28.3** The same victim as in Figure 28.2. (a) The victim cut nude pictures from a magazine and placed photographs of himself on them. (b) The decedent engaged in Pygmalionism, i.e. sex with statues, dolls or mannequins. Dolls with outstretched arms lay near his body. (With kind permission from Wolters Kluwer Health, Inc.: Shields et al. [66].)

behaviour was uncovered when his wife discovered his deceased body in their home.

Older individuals who participate in autoerotic asphyxia may simultaneously engage in transvestism which is exemplified by anal self-stimulation with dildos and self-observation with mirrors or camera [8]. This more extreme autoerotic behaviour is often chosen by experienced asphyxiators who want to enhance their sexual practice.

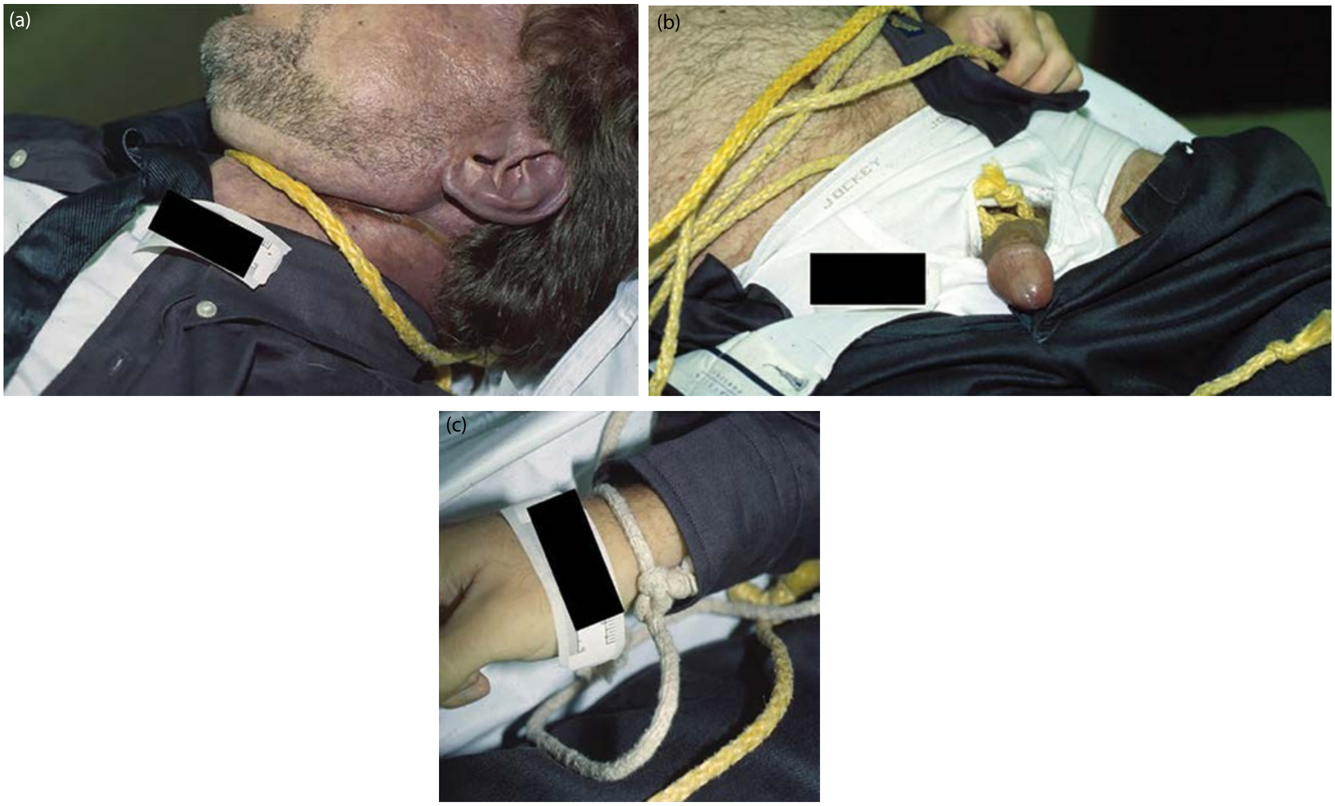
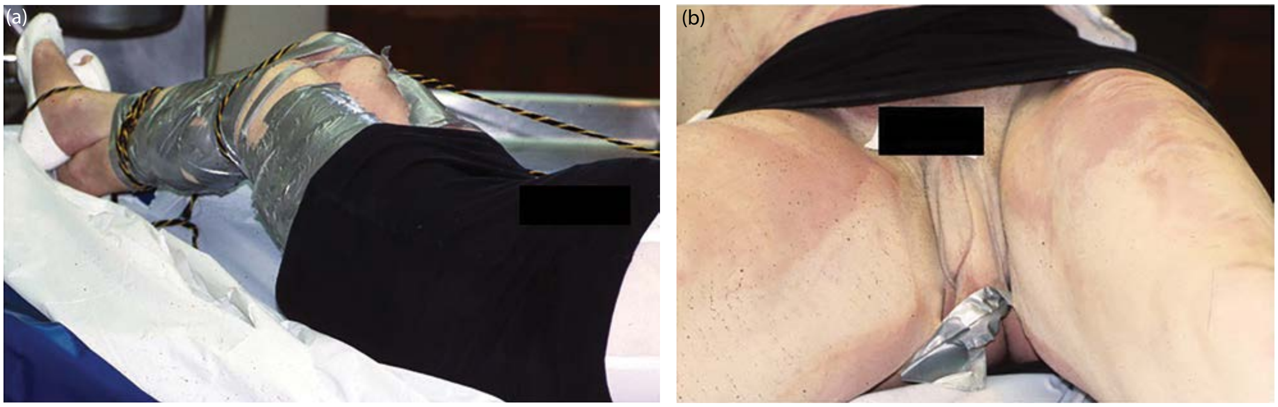


**Figure 28.4** Ligature encircling (a) the neck, (b) the penis and (c) the wrist. (With kind permission from Wolters Kluwer Health, Inc.: Shields et al. [66].)



[redacted]
[redacted]

**Figure 28.5** (a) Duct tape and ligature encircle the legs, and the ligature extends to enwrap the neck. (b) Penis and scrotal sac are bound by duct tape. (With kind permission from Wolters Kluwer Health, Inc.: Shields et al. [66].)

## Paraphilias

A paraphilia (love (*philia*) beyond the usual (*para*)) refers to a disorder in which an unusual act or imagery is necessary to achieve sexual gratification [7–9,49,50,66]. Autoerotic asphyxia has been classified as a paraphilia as the participant yearns for sexual arousal which may only achieved through intense and repetitive sexually arousing fantasies, sexual urges or behaviours involving either a non-human object or the suffering or humiliation of themselves or another human being [31,41]. The ultimate pleasure of autoerotic behaviour is the attainment of hypoxyphilia, which is the craving for a state of oxygen deprivation through either self-induced mechanical or chemical asphyxiation. Participants may experiment with greater thrill-seeking behaviours as they test the limits of their own survival. An unintended fatality may ensue if the release mechanism designed to preserve life fails.

## Manner of death in equivocal cases of autoerotic death

The secretive nature of autoerotic asphyxia coupled with indistinct findings at the death scene in certain cases may prove ambiguous to investigators. As mentioned earlier, females who succumb to autoerotic asphyxia are often found nude with a ligature encircling the neck [12,17]. Sexual devices are usually absent. Awareness of female autoerotic deaths with its lack of typical autoerotic features may be beneficial to forensic investigators [17]. The initial presumptive manner of death is frequently homicide or suicide. A thorough analysis of the autopsy findings, death scene and historical investigation, including an in-depth discussion with the victim's family, may shed light on the manner of death in these complex and baffling cases. Behavioural analysis and reconstruction may also assist in equivocal sexual fatalities [30]. Accidental autoerotic asphyxia fatalities are a distinct entity compared to accidental and suicidal asphyxia deaths and homicidal hanging and strangulation [26].

As autoerotic behaviour is often practised in seclusion and is not intended to be fatal, death due to this act is often shocking to the family and friends of the participant [16]. The death scene may be altered by family members by removing erotic clothing and pornography to conceal the erotic nature of the death with its associated social stigma [12,16]. In addition, forensic investigators do not always adhere to ethical standards when confronted with an autoerotic death [64]. Schoendorff and colleagues [64] described breaches of confidentiality, laxity of work, and an offhand attitude displayed by members of the forensic team in autoerotic death scenes. The determination of the manner of an autoerotic death has important legal, social and economic repercussions that may affect payments of an insurance policy or charge a perpetrator with a homicide [12,48]. Conducting a psychological autopsy, particularly the data-gathering and decision-making process in complex autoerotic cases, is integral to elucidating the accurate manner of death [3,38,45].

Autoerotic death refers to a fatality resulting from a variety of methods to attain heightened sexual arousal. While the majority of autoerotic deaths are accidental, equivocal cases may be deemed to be suicide, natural or homicide [14,37,68]. We investigated 11 cases of 'atypical' autoerotic death that delved into the victim's history and incorporated the complete postmortem examination [68]. Table 28.2 illustrates the causes and manners of death of victims of an 'atypical' autoerotic death [68]. Baber and Bott [4] reported the fatality of a man discovered in an autoerotic setting whose autopsy revealed evidence of pulmonary emboli and lower limb deep vein thrombosis. The manner was natural. Similarly, Cooke et al. [20] also described a natural manner of death in a man with arteriosclerotic and hypertensive heart disease who was using a vacuum cleaner and a hair dryer autoerotically.

**Table 28.2 Causes and manners of death of victims of autoerotic death**

| Accident | Natural | Suicide | Homicide |
|---|---|---|---|
| 1. Asphyxia with plastic bag<br>2. Inhalation of butane<br>3. Inhalation of nitrous oxide within plastic bag<br>4. Electrocution via metal washers on chest<br>5. Electrocution with anal insertion of wire | 6. Ischaemic heart disease | 7. Asphyxia with plastic bag<br>8. Asphyxia with plastic bag<br>9. Gunshot wound to orbit | 10. Asphyxia via duct tape on face and ligature constriction of torso<br>11. Asphyxia via ligature encircling neck |

## Autopsy and toxicological findings

The pathological findings in cases of autoerotic asphyxia are often non-specific and are similar to those encountered in asphyxial deaths [28,66, 76]. Several gross pathological features may be observed singularly or in combination, or may be absent in asphyxial deaths (Table 28.3) [28,66,76]. Investigators have postulated that petechiae result from a mechanical vascular process, specifically impaired or obstructed venous return in spite of persistent arterial input [25,28]. Microscopic evidence of an asphyxial death may include hypoxic/ischaemic damage to tissues if the survival time is delayed. Table 28.4 compares the pathological features of ligature strangulation between suicidal hanging, homicidal strangulation and autoerotic asphyxia [67].

**Table 28.3 Common pathological findings of asphyxial death**

| |
|---|
| Congestion of the face due to venous congestion and poor venous return to the heart |
| Cyanosis resulting from deoxygenated haemoglobin in the venous blood |
| Facial oedema caused by increased venous pressure |
| Petechiae of the eyelids, conjunctivae, sclerae, face and gums due to increased pressure in the microvasculature |
| Injury to skin and soft tissues of the face or neck |
| Internal damage of the neck structures, such as the hyoid bone and larynx |
| Airway obstruction by the tongue in most cases |

Postmortem findings have seldom been reported in autoerotic asphyxia studies. In Walsh and colleagues' analysis of 43 cases of autoerotic asphyxial deaths [76], all victims had evidence of pulmonary oedema, visceral congestion and petechial haemorrhages. Six decedents had sustained fractures of the thyroid cartilage and one had subluxation of a cervical vertebra. In our study of 16 cases of autoerotic asphyxia [66], petechiae of the conjunctivae, skin of the eyelids and extremities, epicardium and pleura were present in 12 victims (75%). No laryngeal fractures or strap muscular haemorrhages were observed. Of the 21 of 43 subjects who underwent postmortem toxicological analysis in Walsh and colleagues' study [76], significant concentrations of alcohol were noted in two cases and a toxic level of barbiturates in one. In our study of 16 victims, the postmortem blood ethanol level was negative in 11 subjects (68.7%) and <0.1% in the remaining five [66]. One decedent had therapeutic blood levels of benzodiazepines and antidepressants.

## Filmed non-lethal autoerotic ligature strangulations and hangings

Sauvageau and colleagues scrutinized non-lethal ligature strangulations and hangings filmed by an autoerotic participant [56,57]. They reported loss of consciousness in 11 seconds, onset of convulsions in 7–11 seconds, and regaining of consciousness in 16–18 seconds in ligature strangulations [57]. Similar early agonal findings were observed in hangings, including decerebrate rigidity observed at 20 seconds in an interrupted hanging [56]. These investigations into a behaviour of a living autoerotic practitioner may elucidate the timing of irreversible brain damage induced by ligature strangulations and hangings.

**Table 28.4 Comparative features of ligature strangulation**

| Characteristic | Suicidal hanging | Homicidal strangulation | Autoerotic asphyxia |
|---|---|---|---|
| Ligature furrow around neck | Inverted V-shaped ligature at the point of suspension | Variability in furrow, often transverse circumferential | Ill-defined or no marking |
| Location | Typically, above the larynx, upward cant | Variably positioned around the larynx | Similar to hanging, with self-release mechanism |
| Ligature force | Full or partial body weight tightens the noose around the neck | Application by force other than body's weight applied to the neck | Similar to hanging, usually with incomplete body suspension |
| Additional neck findings | Internal injuries in less than 1/3 of cases | Bruises, scratch marks to cutaneous neck with internal trauma in approximately 1/3 of cases | Soft ligature with extra padding, internal injuries uncommon |

**Table 28.5 Myths and reality of autoerotic asphyxia**

| Myth | Reality |
|---|---|
| The manner of death may be suicide or homicide. | All autoerotic asphyxial deaths are accidental, by definition. |
| Death can occur with a sexual partner. | All autoerotic asphyxial deaths are solitary activities, by definition. |
| An escape mechanism must be found at the scene. | Often, an escape mechanism is not discovered at the scene. |
| All autoerotic deaths are caused by asphyxia. | Autoerotic death may encompass other methods such as electrocution or drowning. |
| Masturbation is always identified at the scene. | Evidence of masturbation is rare. |

## Conclusion

A fine line exists between the attainment of cerebral hypoxia for sexual pleasure and unconsciousness that may quickly ensue and lead to inevitable death. As autoerotic deaths are innately enigmatic, awareness that this practice may lead to a fatality is critical. Emergency medicine physicians who may encounter participants of autoerotic asphyxia should be educated about the risk factors associated with this behaviour and should be encouraged to identify, report and counsel families and friends coping with autoerotic asphyxia [75].

Several myths pertaining to autoerotic asphyxia persist, and this chapter has strived to dispel these myths (Table 28.5) [55]. The typical autoerotic asphyxia participant acts alone and uses a ligature for the sole purpose of achieving heightened sexual arousal with no intention of ending their life. Variations from the classic autoerotic case may be encountered, depending on the gender of the participant and specific sexual method chosen. These alterations from the typical autoerotic death scene warrant special attention to determine the accurate manner of death. A thorough forensic investigation encompassing a detailed scene inspection, complete postmortem examination and historical exploration may shed light on prior suicidal ideation, covert deviant sexual practices, pornographic material, previous autoerotic behaviour reflected by indentation on rafters of one's residence and intricate bondage entwining the victim.

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# 29 Plastic Bag Asphyxia

*Lisa B.E. Shields and John C. Hunsaker III*

## Introduction

Asphyxia due to emplacement of a plastic bag over the head may result from decreased oxygen concentration or physical obstruction of the mouth and nose [7,32]. It has been postulated that the sympathetic nervous system may be stimulated when a plastic bag is placed over the head which may induce arrhythmias such as ventricular fibrillation [32,51]. This fatal cardiac event may explain the typical absence of postmortem findings in plastic bag asphyxial deaths, including cutaneous and conjunctival petechial haemorrhages, facial congestion, oedema and cyanosis [51,29].

Since the publication of Derek Humphry's book *Final Exit* in 1991 detailing instructions of how to achieve 'self-deliverance' by taking one's own life to escape suffering [23], there has been an increase in the number of plastic bag asphyxial deaths attributed to suicide [23,26,40,41]. Individuals with a debilitating medical illness may select suicide using the methods outlined in *Final Exit*. This chapter discusses the impact of *Final Exit* on the suicide rate, with particular attention to plastic bag asphyxial deaths with or without the supplementation of inert gases. We also delve into the surreptitious act of autoerotic asphyxia where a participant may use a plastic bag to heighten sexual arousal with no intention of causing fatal consequences. This chapter classifies the mechanisms of asphyxia and mentions the findings that may be observed during autopsy and toxicological analysis of plastic bag asphyxia. The death scene may be ambiguous and misleading to investigators of plastic bag asphyxia cases, especially if the evidence has been removed from the scene. A thorough scene analysis, historical investigation and autopsy examination may shed light on the circumstances in equivocal cases of plastic bag asphyxial deaths.

## The impact of *Final Exit*

Published in 1991, Derek Humphry's book *Final Exit* offered people with a terminal illness instructions on how and when to end their lives in a 'bloodless, nonviolent method' [23,24]. *Final Exit* was initially perceived as a controversial 'how to kill yourself' book [26], however, it became number one on the *New York Times* bestseller list within 2 months of its publication [30]. It grew in popularity as individuals embraced the direct message set forth in the book amid the controversy of euthanasia and assisted suicide internationally. Assisted suicide refers to receiving lethal drugs from a physician and swallowing them to cause death [26]. This method of death is legal in only a small number of countries worldwide and in a few states in the United States [14]. Switzerland is the only country that allows foreigners to use medically assisted suicide to die. Self-deliverance denotes taking one's own life to escape suffering and does not involve another person directly [26], although it recommends that a friend or loved one is present during the process of self-deliverance.

From techniques of dying ranging from cyanide ingestion to self-starvation to helium inhalation to carbon monoxide intoxication [10], the step-by-step manual *Final Exit* details how to obtain supplies and pros and cons of different aspects of each method [26]. While this book has been accused of encouraging suicide in individuals with mental disorders, primarily depression [49], adolescents [9] and non-terminal elderly individuals [35], Humphry emphasized that his primary goal for writing the book was to provide a straightforward guide to those suffering from a terminal illness to end their lives by the means of self-deliverance [26]. In 2000, *Supplement to Final Exit: The Latest How-To and Why of Euthanasia/Hastened Death* suggested adding an inert gas such as helium to the plastic bag, due to the ease of obtaining it [25]. The chapter 'A speedier way: Inert gases' details how a person may end their life within 30 minutes by means of a plastic bag and an inert gas such as helium since it may be readily obtained as a helium balloon kit at a toy store. The chapter starts with the disclaimer 'The following words are for information only. This text is not encouraging anyone to take their life.' Humphry recommends taking a sleeping pill or tranquilizer and 'empty[ing] [the] bladder and bowels' prior to the fatal act [26]. To prevent sucking the plastic bag into the nose or mouth, a paper painter's mask may be used.

Humphry founded the Hemlock Society in 1980 which served as an end-of-life care organization, offering information about voluntary euthanasia and moral support without direct assistance [26]. The Hemlock Society's name changed to End of Life Choices in 2003 and subsequently became Compassion & Choices [11]. This latter group works with nationwide and state legislatures, Congress, courts and physicians to support patients' wishes about their end-of-life care.

**Table 29.1 Demographic features of plastic bag asphyxia**

| Study | Years | Number of victims | Age (years) |
|---|---|---|---|
| Byard et al. [7]<br>South Australia<br>2006 | 1984–2004 | 42 (excludes 3 children ages 6 months, 9 years, 11 years) | Mean: 47.1 years (males) (19–88 years)<br>60.5 years (females) (32–89 years) |
| Bullock and Diniz [4]<br>Ontario, Canada<br>2000 | 1993–1997 | 110 | Mean: 60.2 years (16–95 years) |
| Haddix et al. [19]<br>Seattle, WA<br>1996 | 1984–1993 | 53 | Mean: 72.5 years (18–93 years) |
| Jones et al. [32]<br>Southeast Scotland<br>2000 | 1984–1998 | 30 | Mean: 45 years (males)<br>61 years (females)<br>50 years (13–81 years) [Total] |

Marzuk and colleagues [40] investigated the impact of *Final Exit* on the suicide method in New York City in the 2-year period following its publication. The total number of suicides did not increase, but the number of suicides by plastic bag asphyxia increased significantly from 8 to 33 deaths [40]. Furthermore, these authors determined that most of the individuals who consulted *Final Exit* suffered from a psychiatric disorder and were not terminally ill. Marzuk et al. [41] expanded their study to include data from the entire United States in 1990–1991 regarding the influence of *Final Exit*. Similar to the New York City results, suicidal asphyxiations using a plastic bag increased by 30.8 per cent and poisoning increased by 5.4 per cent. The total number of suicides did not escalate over this time period. These authors were unable to determine whether the victims had impulsively committed suicide after reading *Final Exit* or whether the suicidal decision had already been determined and the method was changed due to this book.

## Plastic bag asphyxia: Suicide

While plastic bags are readily available, a paucity of individuals select a plastic bag as a means to end their life. In our study of 2864 suicides in Kentucky between 1993 and 2002, only 16 (0.56%) victims used a plastic bag compared to 1933 (67.5%) victims who utilized a firearm [60]. Plastic bag asphyxia may involve solely a plastic bag or may include inhalation of a gas, such as chloroform [69], ether [3,68], helium [16,17,45,56,58], natural gas (high methane-content mixture) [39], nitrogen [38], butane [1], propane [13,15], exogenous carbon dioxide (dry ice) [13] and toluene [46]. Several studies in the literature described the demographic features of suicidal plastic bag asphyxia (Table 29.1) [4,7,19,32,47,67]. The majority of victims are over the age of 50 years with a female gender preference [4,7,19,47]. The mean age of victims is often substantially higher for females than males [7,32]. The characteristics of a classic case of suicidal plastic bag asphyxia are highlighted in Table 29.2.

In Bullock and Diniz's study, approximately one-third of the victims of plastic bag asphyxia had either attempted suicide or had harmed themselves at least once in their past [4]. Forty per cent of the victims had suffered from a medical illness that decreased their quality of life or was life-threatening. A suicide note was discovered at the scene in half of their cases. An even greater number of victims of plastic bag asphyxia expressed suicidal intent prior to their death in Haddix et al.'s study [19]. A total of 42 decedents (79.2%) had conveyed suicidal intent either in the form of a note (27 cases; 50.9%) or other means (15; 28.3%) [19]. Only 12 victims (22.6%) had previously attempted suicide. In Jones et al.'s study of 27 individuals who committed suicide with a plastic bag [32], 16 victims (59.2%) had a history of a psychiatric illness (depression in 10; 37.0%) and 10 victims (37.0%) had attempted suicide in the past. Literature from the Hemlock Society and/or copies of the book *Final Exit* were rarely found at the scene in these studies [7,19].

We reported two cases of suicidal plastic bag asphyxia in conjunction with helium [58]. In one case, a 69-year-old author in the field of pain management desired 'to end my life on my own terms' after suffering from metastatic colon cancer. In his suicide note, he wrote that he selected suicide 'to shorten the time you have to witness this

**Table 29.2 Characteristics of a classic case of suicidal plastic bag asphyxia**

| |
|---|
| May include inhalation of an inert gas (helium, butane, propane, ether) |
| Age >50 years |
| Female predominance |
| May have suffered from medical or psychiatric illness |
| May have expressed suicidal intent or attempted suicide in the past |
| Plastic bag may not be found |
| Book *Final Exit* may be present |
| Suicide note may be discovered |
| Non-specific findings at autopsy |
| Petechial haemorrhages uncommon at autopsy |
| May have alcohol or drugs in postmortem blood toxicology |

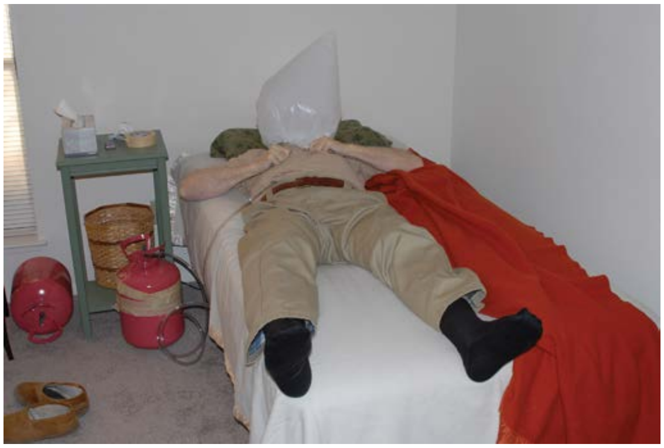

**Figure 29.1** The victim's head is covered with a plastic bag and a tube extends to the helium tank. Two helium tanks were present at the scene. The book *Final Exit* by Humphry [23] was discovered near the body, flagged to the chapter 'A speedier way: Inert gases'. This chapter provides detailed instructions on how to utilize a plastic bag and helium to ensure one's death. (With kind permission from the American Society for Clinical Pathology (ASCP): Shields et al. [58].)

agonizing decline'. He emailed his son-in-law to come several hours later to collect his body. The victim placed a plastic garbage bag over his head and secured it with an elastic scrap (Figure 29.1). A tube extended from a helium tank into the bag. The book *Final Exit* was discovered at the death scene, flagged to the chapter 'A speedier way: Inert gases' (Figure 29.2). In the second case, a 39-year-old man suffering from severe depression was found with a plastic bag over his head and electrical tape encircling his neck (Figure 29.3). A plastic tube from a helium canister extended to the plastic bag. Head space analysis was positive for a volatile substance by gas chromatography (GC).

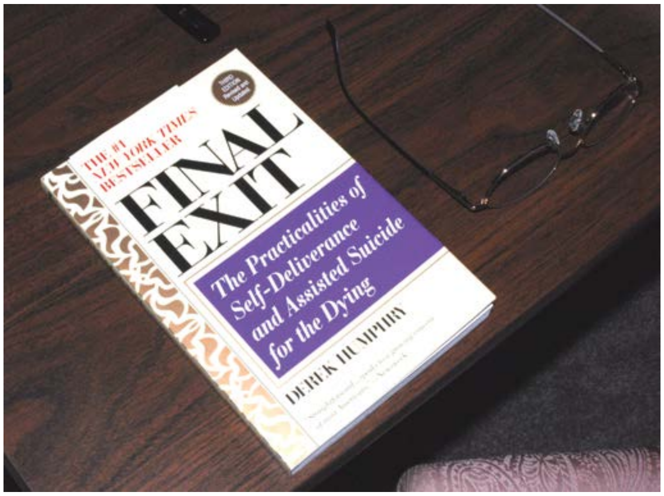
THE #1 NEW YORK TIMES BESTSELLER
FINAL EXIT
The Practicalities of Self-Deliverance and Assisted Suicide for the Dying
DEREK HUMPHRY

**Figure 29.2** The book *Final Exit* by Derek Humphry [5] was found lying next to the victim's body. (With kind permission from the American Society for Clinical Pathology (ASCP): Shields et al. [58].)

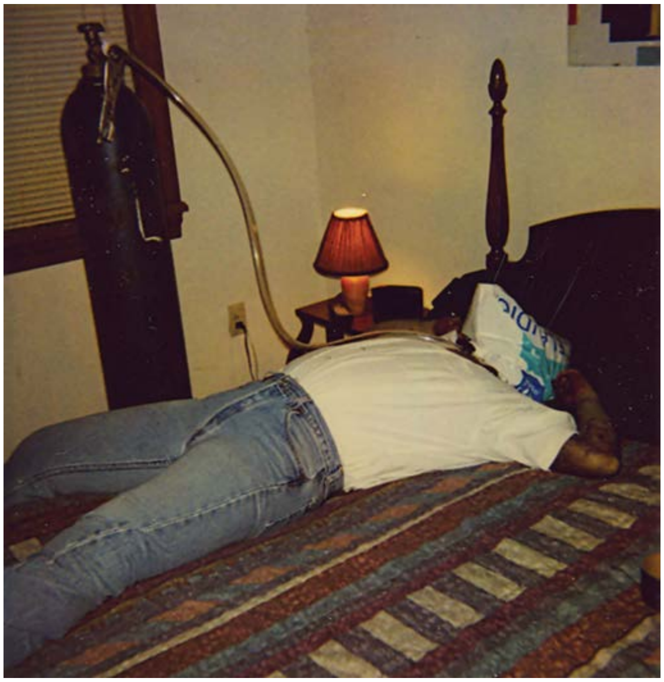

**Figure 29.3** A plastic bag was affixed around the victim's head, and a plastic tube was attached to a helium tank. (With kind permission from the American Society for Clinical Pathology (ASCP): Shields et al. [58].)

## Plastic bag asphyxia: Accident (autoerotic asphyxia)

Autoerotic asphyxia refers to death resulting from failure of a release mechanism of the device, apparatus or prop designed to attain cerebral hypoxia for heightened arousal [6,20,54,59,61]. The 'typical' autoerotic death denotes ligature asphyxiation which encompasses the majority of these cases. The 'atypical' autoerotic death comprises the remainder of asphyxiation mechanisms, including plastic bag asphyxia [31,42,44,55,61,66], inhalation of noxious chemicals such as propane, butane and nitrous oxide [18,27,28,34,37,42,61,62], aqua-eroticum (autoerotic drowning) [22,53,64] and electricity [2,8,33,57,61,63].

Forensic investigators may initially presume that autoerotic asphyxia is suicide. However, evidence at the scene often reveals a nude victim surrounded by pornographic literature, photographs and videos with no intention of ending their life [5]. The decedent may experiment with cross-dressing, and there may be bondage of the extremities and/or penis [59]. The accidental nature of autoerotic asphyxia is often marked by a release mechanism inherent in this practice, frequently a slipknot intended to prevent a fatal outcome [59].

In our study of 'atypical' autoerotic deaths, we described a case of asphyxia with a plastic bag and another of inhalation of nitrous oxide within a plastic bag [61]. Both of these cases were deemed accidental. In the first case, a man was discovered in his bed with a large plastic bag covering his head and rubber banded about his neck. He wore only a brassiere and a wig. There were videotapes at the scene of the victim undressing and applying a Bunsen burner to his perineal area. In the second case, a man was discovered with a plastic bag encircling his head with tubing extending from a bag to a canister containing $N_2O$. He was clad in a camisole, white socks and foam padding resembling breasts. There was evidence of pornographic videos and magazines as well as dildos, a balloon designed for anal insertion and jars of petroleum jelly at the scene. The victim had participated in klismaphilia (arousal from enemas) and Pygmalionism (sex with statues, dolls or mannequins).

## Mechanism of asphyxia

Asphyxial deaths ensue from an inability to uptake or use oxygen in addition to the failure to eliminate carbon dioxide [65]. These deaths are divided into four subsets [65]:

1. Compression of the neck, as in hanging and strangulation.
2. Obstruction of the airway, as in smothering or aspiration of foreign material.
3. Compression of the torso.
4. Exclusion of oxygen by way of depletion and replacement of oxygen by another gas or by chemical interference with oxygen's uptake and utilization by the body.

The last category encompasses situations when oxygen is excluded and carbon dioxide, carbon monoxide, helium or carbon monoxide enter the body. It has been reported that unconsciousness will quickly ensue and death will occur in minutes when an individual breathes with a plastic bag covering the head filled with helium [45].

## Autopsy and toxicological findings

The findings at postmortem examination are non-specific in cases of suicidal plastic bag asphyxia. Humphry describes in *Final Exit* how the death scene may be altered by friends or family members to hide the actual cause and manner of death [26]. Humphry acknowledges that individuals may want to make a statement that their death was a 'rational suicide' by leaving drugs, a plastic bag or *Final Exit* at the scene, but he recommends that a bystander remove the helium gas apparatus as the death will then be attributed to the chronic disease from which the deceased was suffering.

Toxicological analysis does not include testing for helium gas in cases of plastic bag asphyxia with helium. In addition, while petechial haemorrhages are often observed in classic asphyxial deaths [29], they are seldom encountered in plastic bag asphyxia [2,19,32]. In Bollock and Diniz's study [4], only 7 victims (7.5%) had conjunctival or facial petechiae. The most common observations were pulmonary oedema and congestion (6.2%) and visceral petechiae (18.7%). The study by Haddix et al. [19] also described a paucity of petechiae observed upon postmortem external examination. Only six victims had petechiae, and they were few in number (usually less than three in total) and located on the conjunctivae.

Byard et al. [7] noted toxic or lethal levels of prescription medications in 17 cases (37.8%), with benzodiazepines in 10 (22.2%) and alcohol in 8 (17.8%). In addition, 17 victims had histories of depression or had evidence of antidepressant drugs on toxicological analysis. Interestingly, 63 cases (92.6%) had positive results for one or more drugs on toxicological analysis in Bullock and Diniz's study [4]. The most frequently encountered drugs were benzodiazepines (50.0%), diphenhydramine (32.4%) and antidepressants (25.0%). In Jones et al.'s study [32], 11 individuals (36.7%) had alcohol detected in their blood, while 8 (26.7%) had therapeutic blood levels of prescribed drugs. A smaller number of decedents had evidence of ethanol or drugs in the postmortem blood toxicological analysis in Haddix et al.'s study [19], specifically, ethanol in 10 (18.9%) and drugs in 22 (41.5%).

In deaths from gaseous or volatile substances, the lung may yield evidence of toxic substances [52]. The main bronchus is tied off, and the hilum is divided and placed into a nylon bag. It should be sent to the laboratory for airway head space volatile analysis. A plastic bag is not recommended as it is permeable to volatile substances. Glass tubes with an aluminium foil-lined cap or a polytetrofluorethylene (Teflon) liner should be utilized to collect blood samples for volatile substances. Plastic tubes or vials with a rubber septum as a seal are not appropriate to collect blood samples as volatile substances may escape through the rubber.

## Manner of death

A thorough scene investigation, historical analysis and postmortem examination are warranted in cases of plastic bag asphyxia to discern the accurate cause and manner of death. The manner of death may be deemed accidental in cases of autoerotic asphyxia using a plastic bag or when an infant suffocates in a plastic bag, suicidal in terminally ill individuals using a plastic bag asphyxia with or without inert gases, or homicidal when an infant or young child or adult is asphyxiated with a plastic bag by a perpetrator. Homicidal asphyxia may also be disguised as a suicide, which may be deemed natural if the perpetrator removes the plastic bag from their victim after death [50]. In other cases, suicidal asphyxiation may simulate homicide for monetary reasons [12].

Distortion of the death scene by friends or family members may mislead investigators. The decedent's family member may alter the scene in an autoerotic asphyxial death to mitigate the social stigma associated with this type of death [43]. In addition, an individual who selects to end their life by plastic bag asphyxia may decide to leave evidence at the scene such as the plastic bag, drugs, the book *Final Exit* or the helium apparatus (if that was used in the process) or have a witness to their death remove the proof of their means of demise [26]. Interestingly, a plastic bag was present when the investigator arrived in only 36 of 53 victims of plastic bag asphyxia (65%) in Haddix et al.'s study [19].

To further elucidate the manner of death in equivocal deaths (suicide vs accident), a psychological autopsy may prove beneficial [36]. This data-gathering and decision-making process may shed light on psychodynamic factors that may have played a role in the decedent's death, including recent stressors, suicidal ideation and history of depression [36]. The determination of a suicide is based on two factors: the self-inflicted nature of the act and the explicit or implicit intent to die [48]. Explicit intent refers to either verbal or non-verbal expressions of self-injury. A suicide note reflects both explicit intent and the self-inflicted aspect of the suicide. While this type of note provides definitive evidence of suicidal intent, only between 20 per cent and 30 per cent of suicide victims leave suicide notes [21,36].

## Conclusion

While plastic bags are readily accessible, they are uncommonly used in asphyxiation. Deaths due to plastic bag asphyxia may be encountered in suicides when a terminally ill individual yearns to die to escape suffering and in accidental autoerotic asphyxia to achieve heighten sexual gratification and the release mechanism fails. Evidence at the scene may clearly indicate the cause and manner of these deaths, such as a suicide note and the book *Final Exit* in the former or sexual paraphernalia and a slipknot in the latter. The death scene may be misleading or vague when proof of the methods of death has been removed or altered by family members or friends. In addition, the postmortem examination in cases of plastic bag asphyxia often reveals non-specific findings. In this respect, the historical exploration is imperative to conclude an accurate cause and manner of death. The forensic investigator plays a vital role in elucidating the rarity of plastic bag asphyxia by incorporating the evidence uncovered from the scene, autopsy and historical analysis.

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# 30

# Death at High Altitude

*Mattias Kettner*

## Physical background of the hypobaric zone

The atmosphere of Earth is defined by the existence of nitrogen, oxygen and argon as the main constituents of air. It is stratified into layers with the troposphere, reaching from sea level to approximately 12 km above sea level, being the physiological anthroposphere. Above the troposphere, the stratosphere reaches from approximately 12–50 km above sea level, passing into the mesosphere, ranging from approximately 50–80 km above sea level. The next layer of the atmosphere is the thermosphere, ranging from approximately 80–700 km, which comprises the Karman ellipsoid as the boundary of the homo- or anthroposphere at 100 km above sea level [11]. Anthropospheric layers share a comparable chemical composition of nitrogen (≈78%), oxygen (≈21%), argon (≈0.9%). Despite a constant gravitational field of the Earth and subsequently different levels of the constituents in Maxwell–Boltzmann statistics, a relative stability of this composition is accomplished by turbulences and vertical air movements precluding separation based on the constituents' molar masses.

The extent of the troposphere shows considerable seasonal and geographical variation with layer thicknesses ranging from 6–8 km at the poles to up to 18 km at Equator level. This flattened spheroid form results from rotation-based centrifugal forces and the equatorial bulge. The troposphere has a vertical pressure gradient. Barometric pressure at a specific height above sea level can be calculated using the barometric formula (which employs the international standard atmosphere at sea level with an ambient temperature of 15 °C, barometric pressure of 1013.25 hPa, and a temperature gradient of 0.65 K/100 m):

$$ph = 1013.25\left(1 - \frac{0.0065 \cdot \mathrm{h}}{288.15}\right)^{5.225} \text{ hpa}$$

Since the barometric formula uses a standard atmosphere, individual situative variations may vary substantially from this standard setting, resulting in inaccuracy of barometric pressure calculation as compared to pressure measurement.

The thermodynamic equation of the state of ideal gases relates the physical parameters of pressure, volume, temperature and (amount of) substance using the (molar) gas constant. The Boyle–Mariotte law describes the relationship between pressure and volume under isothermal conditions assuming a constant amount of substance. Accordingly, pressure and volume of an ideal gas are reciprocally proportional quantities with a constant product:

$$p1 \cdot v1 = p2 \cdot v2 = v2 = \frac{p1 \cdot v1}{p2}$$

As an approximation, this equation described for ideal gases can be applied for real gases such as gases in anatomical cavities or gases solved in body fluids under normal conditions. In higher altitudes, barometric pressure decreases, leading to adiabatic expansion and cooling of −6.5 K/1000 m within the troposphere. While the surrounding gas (ambient air) increases in volume at higher altitudes, the respiratory volume remains stable, thus leading to a decrease of the amount of substance (oxygen) inhaled per breath. The fraction of inspired oxygen ($F_iO_2$) at sea level of 0.21 decreases to 0.07 at 8000 m height (e.g. in the Himalayan and Karakoram ranges), which leads to a decrease in partial pressure of inspired oxygen.

$$P\mathrm{b} \cdot F_i\mathrm{O}_2 = P_i\mathrm{O}_2(\mathrm{Torr})$$

Furthermore, the equatorial bulge and the concomitant spheroid ellipse form of the troposphere cause differences in barometric pressure at the same height above sea level subject to the measured region of Earth. Partial pressures of inspired oxygen in areas close to the poles are significantly lower than those measured in areas close to the Equator. e.g. $PiO_2$ of mountaineers ascending to Mount Mc Kinley-Denali (height 6190 m; 63° northern latitude) closely resemble those of mountaineers ascending to Mount Everest (height 8848 m; 27° northern latitude)[37].

## Barotrauma at high altitude: Hypobaric barotrauma

In a broader sense, the term 'barotrauma' characterizes a pathological condition of the human body evoked by a change in ambient air pressure and concomitant reductive or expansive gas volume adaptations. For pressure equalization within tolerable limits, the human body can utilize compensatory mechanisms such as anatomical connections (e.g. of nasopharyngeal cavities) or increase of the breathing rate. Harmful effects on the human body are due to rapid changes within moderate and potentially tolerable limits, changes exceeding tolerable limits, long-time exposure to high altitude or pathological conditions of anatomical structures involved in pressure equalization. Thus, acute altitude illnesses can be distinguished from chronic illnesses as a result of long-time or lifetime exposure to high altitude.

## Acute altitude illness

Acute altitude illnesses (AAIs) (acute hypobaric traumata) are seen predominantly in patients with a prior rapid ascent to higher altitude (e.g. as tourists and mountaineers). Although physiological changes and acute illness may appear in susceptible individuals beginning at a height of approximately 1500 m above sea level, the term 'high altitude' is usually defined as heights above 2500 m [20]. Symptoms of acute high-altitude illness may appear up to 5 days after a rapid ascent to high altitude (acute exposure) and can be grouped into three different forms: Acute mountain sickness, high-altitude cerebral oedema and high-altitude pulmonary oedema [25].

### Acute mountain sickness

Acute mountain sickness (AMS) is a syndrome seen in unacclimatized patients after ascent to high altitude. The rather non-specific symptoms include high-altitude headache (HAH, which may be absent in about 5 per cent of the patients with AMS [32]) and in addition nausea, vomiting, anorexia, insomnia, dizziness, obnubilation, fatigue or a combination of the latter symptoms [26]. HAH is a common finding in patients with rapid ascent to high and very high altitudes. It is defined as a headache presenting after rapid ascent to altitudes of >2500 m, which resolves within 24 hours after descent. Risk factors to develop HAH include low oxygen saturation, reduced fluid intake, exertion and a medical history of migraine [26].

Symptoms of AMS usually occur with a 4–12 hour delay after gain in altitude and regress over 1–3 days. Progression of symptoms including nausea and headache not responding to first-line anti-emetics and analgesics may indicate progression from AMS to high-altitude cerebral oedema (HACE) [25,41]. The severity of AMS in a presenting patient can be assessed using the Lake Louise Score (LLS) for the diagnosis of AMS [35] (Table 30.1). Here, given a suitable history of ascent to high altitude, a score based on a short self-reported questionnaire can be complemented by a clinical assessment score and a functional score. A score of 3 or more in the self-reported questionnaire constitutes AMS.

### High-altitude pulmonary oedema

High-altitude pulmonary oedema (HAPE) is a potentially lethal form of altitude sickness typically resulting from rapid ascent to high altitude by unacclimatized visitors to high-altitude regions or from well-acclimatized high-altitude residents returning from low altitudes (re-entry HAPE) [25]. Oedema formation is attributed to (severe) pulmonary hypertension resulting from vasoconstriction due to hypobaric hypoxia [27]. A comprehensible explanation for the characteristically patchy oedema (Figure 30.1) is an uneven hypoxic vasoconstriction leading to a situation where unconstricted vessels fail due to massive pressure exposition [40].

Clinically, HAPE is characterized by an increasing breathlessness accompanied by one or a combination of the above-mentioned symptoms of AMS beginning a few hours after arrival in high altitude. An initially dry cough turns productive with frothy white and later blood-tingled sputum [40]. Upon auscultation, crackles may be heard at the lung bases. In addition to tachycardia and tachypnoea, cyanosis and mild pyrexia may develop [25,38]. Reported incidences of HAPE vary substantially (due to varying subject populations, final altitude and rapidity of ascent) and range from 0.57 per cent [28] to 10 per cent [4]. Rapid deterioration and potentially death may be seen in those HAPE patients who do not descend or receive medical treatment. Optimal treatment depends on individual access to treatment facilities. HAPE patients should descend if there is no treatment possibility of receiving supplemental oxygen, entering a hyperbaric chamber and receiving a pulmonary vasodilator [25]. Furthermore, phosphodiesterase inhibitors may be useful, while diuretics are contraindicated [38].

### High-altitude cerebral oedema

High-altitude cerebral oedema (HACE) has been defined as a condition occurring in persons who have recently arrived at high altitude, usually secondary to AMS or HAPE, and marked by disturbances of consciousness potentially progressing to deep coma, psychiatric changes of varying degree, confusion and ataxia of gait [16]. It is mostly seen as an *aggravatio per continuitatem* of severe AMS and occurs commonly with HAPE. As in HAPE, reported incidences vary substantially and have been described (conceding obvious differences in study designs, ascent rates and data acquisition) ranging from 0.5 per cent (varying rates of ascent in 5355 visitors to 4555 m in Tibet) [6] to 31 per cent (Vedic pilgrims at 4300 m in Nepal) [7]. HACE most commonly develops over 24–48 hours after initial symptoms of AMS have been detected. Typically, changes in consciousness with drowsiness, progressing lassitude and evident confusion are accompanied by ataxic gait [16]. The initial state of HACE has been compared to the state of mild drunkenness [8]. Patients presenting symptoms of HACE necessitating hospitalization should receive a complete evaluation including a complete history as well as a physical and laboratory examinations including serum electrolytes, blood cell count and renal function. Examination of cerebrospinal fluid may be used to rule out central nervous system (CNS) infections. CNS imaging

**Table 30.1** **Lake Louise scoring system of acute mountain sickness (AMS). A sum of 3 or more (headache and at least one other symptom must be present) is taken as AMS in the self-report questionnaire. Clinical assessment of mental status, ataxia, and peripheral oedema requires examination by an experienced observer. A combined (self-reported and observer-based) sum of >5 has been suggested for the diagnosis of AMS [5]. The closing question concerning functional consequences has later been added to the LLS system to enable assessment of overall functional limitation**

| Self-report questionnaire | | Score Points |
|---|---|---|
| Headache | No headache | 0 |
| | Mild headache | 1 |
| | Moderate headache | 2 |
| | Severe, incapacitating headache | 3 |
| Gastrointestinal symptoms | No gastrointestinal symptoms | 0 |
| | Poor appetite/nausea | 1 |
| | Moderate nausea/vomiting | 2 |
| | Severe nausea/vomiting, incapacitating | 3 |
| Fatigue and/or weakness | Not tired or weak | 0 |
| | Mild fatigue/weakness | 1 |
| | Moderate fatigue/weakness | 2 |
| | Severe fatigue/weakness, incapacitating | 3 |
| Dizziness/lighheadedness | Not dizzy | 0 |
| | Mild dizziness | 1 |
| | Moderate dizziness | 2 |
| | Severe dizziness, incapacitating | 3 |
| Difficulty sleeping | Slept well as usual | 0 |
| | Did not sleep as well as usual | 1 |
| | Woke many times, poor night's sleep | 2 |
| | Could not sleep at all | 3 |
| Symptom Score: | | ___________ |
| Clinical assessment | | |
| Change in mental status | No change in mental status | 0 |
| | Lethargy/lassitude | 1 |
| | Disoriented/confused | 2 |
| | Stupor/semiconsciousness | 3 |
| | Coma | 4 |
| Ataxia (heel to toe walking) | No ataxia | 0 |
| | Manoeuvers to maintain balance | 1 |
| | Steps off line | 2 |
| | Falls down | 3 |
| | Can't stand | 4 |
| Peripheral oedema | No peripheral oedema | 1 |
| | Peripheral oedema at one location | 1 |
| | Peripheral oedema at two or more locations | 2 |
| Clinical Assessment Score: | | ___________ |
| Total Score: | | ___________ |
| Functional Score | | |
| Overall affection of activity | No reduction in activity | 0 |
| | Mild reduction in activity | 1 |
| | Moderate reduction in activity | 2 |
| | Severe reduction in activity, e.g. bedrest | 3 |

using magnetic resonance (MR) tomography (e.g. FLAIR, DWI, SWI) may be helpful if available.

Ethiologically, ascent-related hypoxia causes an overall increase in cerebral blood flow (CBF) (despite the opposing effect of hypocapnia-caused cerebral vasoconstriction). Even though there is no clear correlation between increased CBF and HACE (or AMS), MR studies suggest a vasogenic origin of the oedema with increased permeability of the

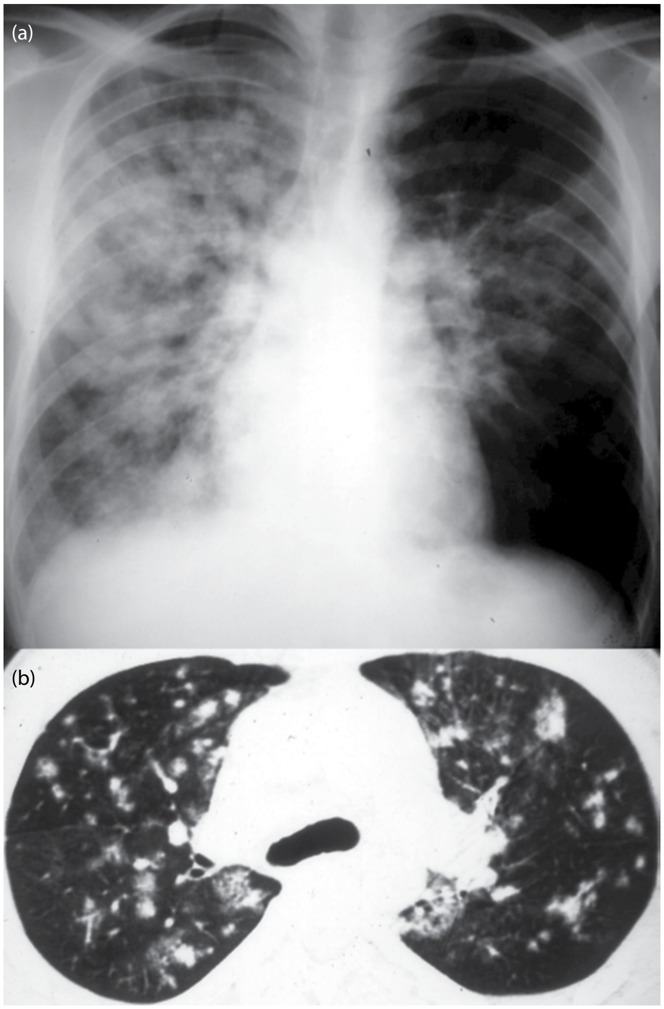

**Figure 30.1** (a) x-ray of a 37-year-old male HAPE patient with patchy to confluent oedema distribution. (b) Computed tomography section of a 27-year-old male patient with recurrent HAPE showing the typical patchy distribution of oedema. (Courtesy of Professor Bärtsch.)

vascular endothelium [40] with increased signal in the white matter, markedly in the splenia of the corpora callosa [17]. Furthermore, hypoxia-induced cellular ion pump failure leads to an increase of intracellular sodium and thus osmolarity with subsequent influx of water, resulting in cellular swelling in the sense of a cytotoxic oedema [14,40].

In patients diagnosed with HACE, rapid descent to lower altitudes should be pursued as the primary therapeutic measurement. Dexamethasone has been proven to help the cerebral symptoms [12], but it has little or no effect on the physiological alterations [24]. In a pre-hospital setting, oxygen therapy and hyperbaric bags may be tried. Once the hospital has been reached, standard intracerebral oedema therapy can be applied [40].

## Mortality, autopsy results and management in acute altitude illness

Whereas numerous studies have been carried out regarding mountain mortality with mortality rates for specific routes of regions of interest as well as specific recreational activities, studies on AAI fatalities and especially on autopsy findings in AAI are rather scarce and usually display a limited number of cases. Possible reasons therefore may be the rare nature of fatalities due to AAI, incomplete collection of autopsy data (as expeditions are frequently multinational undertakings), missing postmortem examinations (due to internationally varying legal requirements), as well as missing bodies precluding autopsy (e.g. accounting for 20% of all fatalities above base camp on Mount Everest 1921–2006) [13].

Given a suitable case history (death in or after a visit to high altitude, anamnesis with AMS/HAPE/HACE symptoms), an AAI-related cause of death should be taken into consideration. In an autopsy study of 10 HAPE-associated cases, autoptical examination revealed severe diffuse oedema in all cases with increased organ weights of the lungs (mean 1682 g, weights ranging from 1200–3000 g). Further gross anatomical findings included airways filled with fluid (frequently blood-stained) and dilation of the right ventricle and atrium. Cerebral oedema seen in some of the cases was attributed to hypoxia of HAPE rather than being an etiologic factor [19]. These gross anatomical findings were comparable to those of earlier studies [2]. Autopsy findings in HACE include macroscopic aspects of cerebral oedema (e.g. cerebellar herniation and flattening of gyri) as well as white matter spongiosis and petechial haemorrhage of the brain [9,33]. Fundoscopic inspection may reveal papilloedema as well as retinal haemorrhage [16].

## Chronic altitude illness

Chronic conditions resulting from high-altitude dwelling affect approximately 140 million inhabitants of geographical regions at more than 2500 m above sea level. People living in these regions (either as sojourners, spending years at high altitude for economic reasons, or as permanent residents), mainly the Andes, the Himalayas and Tibet, and the Ethiopian highlands (but also parts of the Rocky Mountains or potentially any other populated high-altitude area), live in a state of constant alveolar hypoxia and frequently exhibit severe hypoxemia resulting in concomitant pathological alterations [39]. Since numerous terms for these conditions were used in the past, the International Society for Mountain Medicine issued a consensus statement in 2005, following which the terms 'chronic mountain sickness' and 'high-altitude pulmonary hypertension' are used [23].

### Chronic mountain sickness (Monge's disease)

In 1925, Carlos Monge Medrano reported the first case of polycythaemia in a Peruvian patient living at Cerro de Pasco [29], giving this entity its name. Following this case report, a series of patients have been reported from South America but also from high-altitude regions in the United States of America and Asia (for details see [39]).

Chronic mountain sickness (CMS) is defined in the 2005 ISMM consensus statement [23] as:

> 'a clinical syndrome that occurs in natives and long-life residents above 2500 m. It is characterized by excessive erythrocytosis (Hb $\geq$19 dl$^{-1}$ for females and $\geq$21 dl$^{-1}$ for males), severe hypoxaemia and in some cases moderate or severe pulmonary hypertension, which may evolve into cor pulmonale, leading to congestive heart failure. The clinical picture of CMS gradually disappears after descending to low altitude and reappears after returning to high altitude.'

Excessive erythrocytosis may reach haematocrits of up to 91 per cent [21], causing an increase in blood viscosity which in turn leads to a decrease in CBF [36].

CMS patients typically exhibit neuropsychological complaints including headache, fatigue, somnolence and depression. Patients are apt to gain weight and display poor exercise tolerance. Characteristically, these symptoms disappear after descent to sea level to reappear after return to high altitude [40]. Macroscopically, a combination of 'virtually black lips and wine red mucosal surfaces' has been described in Andean natives [18], whereas signs in Caucasians may be limited to those of hypoxic lung disease at sea level (e.g. clubbed fingertips and congested conjunctivae) [40].

The best therapeutic strategy is avoiding high-altitude habitation, i.e. rapid and permanent descent to lower altitude. If this is not possible, venesection is an effective therapeutic option. Furthermore, acetazolamide and medroxyprogesterone have been suggested for long-term treatment [22,31].

## High-altitude pulmonary hypertension

The Euler–Liljestrand mechanism describes the effect of pulmonary vasoconstriction as a result of hypoxia in cats [10] and is thus the physiological basis for the understanding of high-altitude pulmonary hypertension (HAPH). HAPH is the result of long-term hypoxia under high-altitude living conditions leading to global lung vasoconstriction as opposed to the beneficial effects of this mechanism in other pathological conditions (e.g. pneumonia), when blood flow is diverted to other, well-ventilated areas of the lung.

HAPH affects sojourners to high altitude as well as high-altitude dwellers. Patients in a study comprising 21 military members with long-term deployment to >5000 m of altitude presented with dyspnoea, cough and effort angina as signs of dependent oedema. Upon examination after descent to low altitude they displayed cardiomegaly with right ventricular enlargement and in 17 cases pericardial effusion. Pulmonary artery pressure was elevated at rest and under mild exercise [1]. In addition, in a Lhasa-based study of 17 patients, liver enlargement and increased jugular venous pressure were noted [30]. In a study comparing Han Chinese and Tibetan populations in Tibet, clinical signs described included cyanosis, oedema of the face, crackles of the chest, tachycardia, tachypnoea and liver enlargement. In this study, up to 3.64 per cent of children examined in the Han population suffered from HAPH [15].

## Mortality, autopsy results and management in CMS/HAPH

Reports on CMS/HAPH fatalities with autoptical evaluation of relevant pathologies are scarce. In a 1973 publication two earlier reports were reviewed and four cases of CMS/HAPH patients (the report from1973 distinguishes between three types of chronic mountain disease not congruent with the 2005 ISMM consensus statement) with autoptical evaluation were presented [3]. All patients in these publications died of (severe) cardiac insufficiency showing right (and in one case left) ventricular hypertrophy and peripheral pulmonary arterial thickening. Furthermore, two patients were described as obese, the other two had scoliosis, both conditions aggravating CMS pathology.

Reports on infant fatalities in the Han Chinese and Tibetan population in Tibet have characterized postmortem findings as extreme medial hypertrophy of muscular pulmonary arteries and muscularization of pulmonary arterioles. Massive hypertrophy and dilatation of the right ventricle and the pulmonary trunk were seen macroscopically [34].

Given a suitable case history of long-term dwelling in high altitude and a patient history presenting the above-mentioned CMS symptoms, a CMS fatality should be taken into consideration. Upon autopsy, right ventricular hypertrophy and hypertrophy of the pulmonary trunk, pericardial effusion, liver enlargement as well as histologically proven (peripheral) pulmonary arterial thickening may be expected alongside corresponding haemoglobin and haematocrit values. External inspection may reveal cyanosis and oedema of the face as rather unspecific symptoms.

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# 31 Death at Depth

*Mattias Kettner*

Diving activities can be mainly grouped into two different categories, professional diving and recreational diving. Professional divers are usually well trained for the specific tasks they fulfil and are subject to periodic medical check-ups. Diving-related medical conditions or fatalities in this group usually originate from previously non-limiting (not detected) pre-existing medical conditions, gear-related problems or wrong assessments of work-related diving situations. The group of recreational divers has constantly grown over the past decades, with the Professional Association of Diving Instructors (PADI), the scuba diving certification agency with the largest number of certifications per year, claiming to have certified an average of more than 900 000 divers each year for the last 20 years. This gives a perspective on potential recreational diver numbers. Although a medical check-up is required to become certified, regular medical check-ups are advised but are usually not rigorously enforced. Furthermore, since recreational divers are often diving only once a year or less, it is frequently not seen as an inherent necessity.

Recreational diving is certainly most common at classical coastal diving spots, but there are also numerous diving spots at inland lakes, quarries and even flooded mines. With the accessibility of these and faraway diving spots being quick and easy, clinical physicians as well as forensic medical experts are nowadays more likely to encounter disorders and injuries acquired during diving or fatalities as a result of the activity.

A dive is divided into at least three stages that imply distinct potential for pathological medical conditions and thus the forensic medical experts' attention. While descending to depth, the compression phase may lead to under-pressure in gas-filled cavities when pressure equalization is insufficient. After reaching the desired depth with then nearly constant ambient pressure, inadequate personal gear (inadequate thermal insulation, buoyancy problems, diving gear malfunction) may lead to hyper- or hypothermia, inadvertent ascent/descent, or breathing and coordination problems under water. While ascending, decompression must be controlled, with additional decompression stops dependent on the depth. In this phase, uncontrolled or overhasty ascent may lead to overpressure-related medical conditions. This chapter is intended to familiarize the reader with common diving physics and dive-related medical conditions.

## Physical background of the hyperbaric zone

The same physical principles apply for the hyperbaric zone as were seen for the hypobaric zone (see Chapter 30). In diving, the human body and its gas-filled cavities are subject to a combined pressure resulting from the barometric pressure at sea level and the pressure exerted by the water column between the body and the sea surface. Due to the density differences between air and water, the pressure changes experienced by divers over a specific depth change are far greater than those experienced by mountaineers as they ascend a mountain. The effective pressure can be expressed either relative to a vacuum as absolute pressure (1 bar at sea level, 2 bar at 10 m of depth under sea surface level (usl)) or relative to the sea surface level as gauge pressure (0 bar at sea surface level, 1 bar at 10 m of depth usl). The Boyle–Mariotte law states that, given a constant amount of gas and a constant temperature, the absolute pressure exerted by a given mass of an ideal gas is inversely proportional to its volume. For example, a balloon transferred from sea surface level to 10 m usl (with a duplication of absolute pressure from 1 bar to 2 bar) will have half of its sea surface level volume.

Dalton's law of partial pressures applies as total pressure exerted in a mixture of non-reacting gases is equal to the sum of partial pressures of each of the gases, which is shown by an increase of, for example, the inspiratory partial pressure of oxygen $P_iO_2$ of 0.21 at sea surface level to 0.63 in 20 m usl and of $P_iN_2$ from 0.79 to 2.37 respectively.

According to Henry's law, this equalization is also true for the dissolubility of the gas components (constituents of air and other gas components due to intended and unintended technical manipulation) regarding bordering liquids (e.g. body fluids and tissues). This is because the amount of gas dissolved in a liquid is proportional to the partial pressure of the gas over the liquid given a constant temperature. In diving, the crucial factor in these equalization processes is the time elapsed per metre depth change, which is the reason for decompression stops at calculated depths depending on maximum diving depth and personal constitution.

Diving-related medical conditions, which are not based on pre-existing health problems (e.g. stable coronary artery disease with exercise-related exacerbation leading to myocardial infarction during a dive), can be grouped into two categories: Barotrauma and decompression sickness.

## Barotrauma

In a broad sense, barotrauma is a medical condition of the human body effected through a change of pressure and the concomitant (either reductive or expansive) volumetric changes of gas-filled cavities of the body. In a narrower sense (and for the following text), the term 'barotraumata' will be used to depict hyperbaric barotraumata under water. Occurrence of damage is crucially dependent on the pace of pressure changes. In contrast to inanimate objects with elastic material properties (e.g. a balloon), the human body (and its gas-filled cavities) disposes of compensatory mechanisms such as anatomic interconnections of gas-filled cavities to the respiratory tract (e.g. paranasal sinuses, middle ear) and is thus capable of at least some pressure change compensation via breathability of its lungs, if changes occur with modest pace. Accordingly, barotraumata are usually the result of rapid changes between pressure levels (e.g. rapid ascent due to ignorance concerning diving technique or to emergencies), or they occur as the result of pathological changes of involved anatomical structures precluding pressure equalization, such as occlusion of the Eustachian tube or carious cavity formation in teeth.

### Pulmonary barotrauma

Although the lung may tolerate different pressure levels well (e.g. in saturation diving), the rate of pressure equalization at a rapid ascent (emergency situation) or descent (free- or apnoea diving) is limited. Pulmonary barotraumata (PBT) mainly occur during decompression upon ascent, and are called PBT of ascent. Injury occurs due to ascent-related volume distension of lung tissue (when gas cannot be vented adequately) with rupture of alveoli as well as development of a harmful transmural pressure gradient [6]. Gas in the lung interstitial space may enter the mediastinum (causing mediastinal emphysema) as well as the pleural space (causing pneumothorax) [15]. Interstitial gas dissection along the oesophagus may lead to pneumoperitoneum and, alongside the cervical tissue, it may lead to dysphagia, hoarseness, throat pain and neck emphysema [1] as well as chest emphysema. Upon arrival at the sea surface, explosive exhalation as well as a characteristic sudden high-pitched cry has been described [6]. Clinical symptoms include those typical for (tension) pneumothoraces such as dyspnoea, tachypnoea, (pleuritic) pain, cough as well as haemoptysis. Upon auscultation, the so-called Hamman's crunch may be heard as a sign of mediastinal emphysema [10]. Chest X-ray may show the tram-track sign as a result of air stripping the pleura from the edge of the cardiac shadow [6] (Figure 31.1).

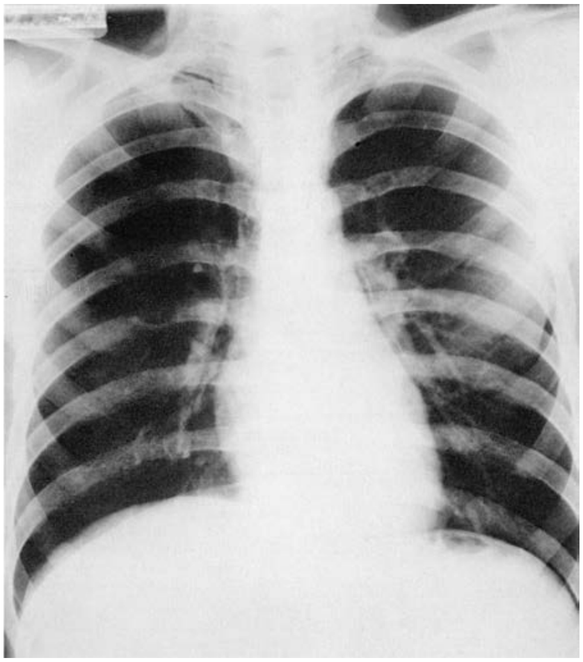

**Figure 31.1** Chest X-ray of pulmonary barotrauma of ascent showing the so-called tram-track sign caused by mediastinal emphysema. (From Edmonds C et al. In: *Diving and Subaquatic Medicine*. 5th ed. CRC Press, 2016, pp 65–79.)

Arterial gas embolism (AGE) may result from high alveolar pressure forcing gas into alveolar capillaries [1] or be due to entry into vessels affected simultaneously while alveoli rupture during distension [6]. Airlock of the left ventricle of the heart is reported to be relatively uncommon [6], with approximately 5 per cent of AGE-affected divers showing cardiac arrest after apnoea and loss of consciousness due to filling of cardiac chambers or great vessels with air [1]. Gas bubbles tend to distribute with the flow contingent upon factors such as position, buoyancy and movements of the diver. In an ascending position, this distribution mainly affects the upper body parts, and particularly the brain. Cerebral arterial gas embolism (CAGE) is the most feared complication of PBT with hypoxia leading to loss of consciousness usually within minutes after surfacing. Further symptoms include hemiplegia, stupor and confusion, visual disturbances, seizures, vertigo and headache [1].

PBT due to an increase in ambient pressure (also termed inverse barotraumata) have been reported in freediving (after reaching the residual volume at about 30 usl equivalent to 3 bar gauge pressure leading to a reduction in total lung capacity from, for example, 6 litres to 1.5 litres), in regulators with high respiratory resistance and while operating with overlong snorkels. After exhausting compensatory mechanisms, increased under-pressure in the pleural cavities leads to pulmonary oedema and finally alveolar rupture with haemorrhage. Cognizant of these limitations, deep dives in freediving may be explained by application of special breathing techniques, training of auxiliary respiratory muscles and long-time habituation.

## Ear barotrauma

Ear barotraumata (also called otological or aural barotraumata) may affect the external ear, the middle ear and the inner ear.

External ear barotraumata are seen in patients with either tight-fitting suit hoods or with occlusion of the external auditory canal (e.g. being blocked by cerumen or foreign bodies). Symptoms include pain in the affected ear, swelling, erythema, petechial haemorrhages and blood-filled cutaneous blebs [8].

Middle ear barotrauma (also called barotitis media or middle ear squeeze) is the most common diving-related medical disorder. It occurs when the Eustachian tube cannot be opened for pressure equalization via the Valsalva manoeuvre, for example because of an acute medical condition. Symptoms include depth-dependent otalgia, vertigo, middle ear oedema, transudation, haemotympanum and tympanic membrane rupture. Late complications may include chronic tympanic membrane perforation and chronic otitis media [8].

When pressure equalization is not equivalent in the bilateral middle ear spaces (threshold of 45 mmHg pressure difference), patients may show nausea, vomiting, disorientation and general malaise. This condition is based on disturbed labyrinthine function and is called alternobaric vertigo [8,13].

Inner ear barotrauma includes perilymph fistula, intra-labyrinthine membrane tear, and inner ear haemorrhage [7]. Divers suffering from inner ear barotrauma show sensorineural hearing loss, loud roaring tinnitus and persistent vertigo after diving [8]. While cochlear injury is reported to be permanent in about half of the cases, vestibular symptoms are usually temporary [6].

## Dental barotrauma

Dental barotrauma ranges from the initial symptom of pain to manifest destruction of teeth. In a study focusing on dental barotraumas, 15 per cent of the included divers reported having suffered dental barotrauma in the past, with tooth injuries/fractures (also called odontocrexis) found in 6.3 per cent [19]. Based on the underlying pathomechanism, dental barotrauma may be subdivided into three groups.

The first group is in divers with an untreated or not sufficiently treated carious tooth with cavity formation and a thin cementum remnant allowing for implosion upon descent and explosion upon ascent. The second group is of divers with gas space formation as a result of either medical conditions allowing for infection in the surroundings of roots, nerves, pulp and as a result of poorly inserted fillings or other dental work, and those with secondary erosion. While destruction of the involved teeth and/or dental work is rare, gas-filled spaces may fill up with blood and effusion upon descent, causing considerable pain. On ascent, pain may be absent, potentially evoked by distension of entrapped gas remnants. In a third group, divers with preceding oral surgery or dental work may suffer from surrounding tissue emphysema caused by the surgical interruption of the mucosa [6].

## Equipment barotrauma

Equipment barotrauma reflects the physical properties of diving gear and the human body focusing on the interjacent gas-filled space.

Facial barotrauma of descent (also called mask squeeze) is typically seen in divers with either goggles (as erythema and oedema of the skin surrounding the eyes (also called ocular purpura or purpura gogglorum [12,17]) or with insufficient pressure equalization (e.g. through nasal exhalation). Clinically, facial barotrauma ranges from simple erythema to skin, conjunctival and orbital haemorrhage [6].

Suit barotrauma may occur during descent and ascent. During descent, skin may be sucked into folds of drysuits or poorly fitting wetsuits resulting in skin marks, which are usually painless. Respective problems may arise from the usage of a P-valve with a genital squeeze as well as usage of a she-P-valve with a squeeze component and subsequent urinary infection [11]. Suit barotrauma during ascent is found mainly in divers using standard diving suits. While ascending, gas constantly has to be discharged from this type of suit. If for any reason this discharge does not take place, the diver is exposed to barotrauma of ascent, decompression sickness, imprisonment and physical trauma [6]. Head and body barotrauma of descent (also called divers' squeeze) is found in standard diving using rigid helmets. Upon descent, diver and suit are forced into the helmet. Depending on the rate of descent and the total depth, the potentially lethal symptoms range from dyspnoea to oedema and haemorrhage of the skin, mucosa and internal organs (e.g. brain, heart and lungs) as well as fractures (e.g. of the clavicles).

## Gastrointestinal barotrauma and pneumoperitoneum

Gastrointestinal barotrauma, usually presenting as gastric rupture, is a rare condition, in which rapid ascent leads to distension of entrapped gas volumes in the stomach. Sharp abdominal pain after a rapid ascent is the cardinal symptom, and surgical intervention is the essential therapeutic strategy. Pneumoperitoneum as the result of gas emission into the peritoneal cavity can be seen upon radiological examination [14,18] and may lead to an abdominal compartment syndrome inducing respiratory, cardiovascular and renal compromise [3].

## Other barotraumata

Pneumocephalus due to diving is a rare medical condition. Air enters the cranial cavity through a communicative fistula, which may be caused by descent barotrauma of

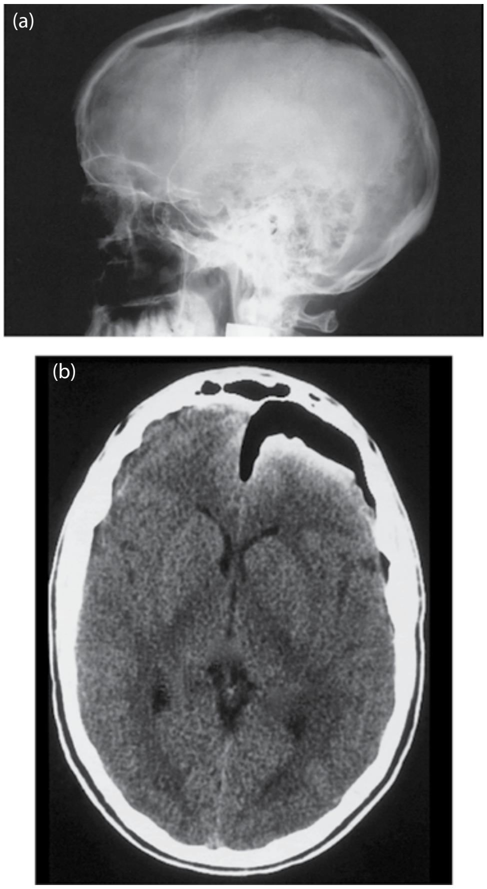

**Figure 31.2** Sinus barotrauma of ascent inducing pneumocephalus. (a) Lateral view X-ray of the vertex with crescent-shaped pneumocephalus; (b) computed tomography section of anteriorly located pneumocephalus. (From Edmonds C et al. In: *Diving and Subaquatic Medicine*. 5th ed. CRC Press, 2016, pp 65–79.)

paranasal sinuses, for example (Figure 31.2). Clinically, symptoms range from headache upon ascent to severe and potentially lethal brain injury and cranial nerve lesions [2,6].

Intraosseous pneumatocysts may lead to pain during descent and ascent due to compression and concomitant haemorrhage and ascent-related distension respectively.

## Decompression sickness

The term 'decompression sickness' (DCS) comprises a broad variety of symptoms and clinical manifestations, which are due to gas bubble formation of previously dissolved gas upon ascent-related partial pressure equalization (following Henry's law). During descent, increasing partial pressures of gases lead to progressive dissolution of gases in body fluids and tissues. Nitrogen as the main constituent of air is five times more soluble in fatty tissue than in blood or water. When the diver ascends and partial pressures decrease, partial pressure equalization of the metabolically inert gas nitrogen is dependent on perfusion, which is lower in fatty tissue [5]. The combination of high nitrogen content and low perfusion of fatty tissues may then lead to supersaturation of tissues and insufficient equalization resulting in bubble formation during rapid ascent. Dependent on the extent of bubble formation and their localization, bubbles may either cause DCS or remain unobtrusive as so-called silent bubbles.

Based on its clinical manifestations DCS is subdivided into DCS type I and type II.

### DCS type I

DCS type I (also called 'the bends' or 'pain-only DCS') is the most common form of DCS, representing 60–75 per cent of all cases. It is characterized by joint pain, predominantly seen in knee, elbow and shoulder joints, attributed to extravascular gas bubbles causing mechanical distorsion [5].

Skin manifestations of DCS may vary and include erythematous rash and pruritus with erysipelas rash and cutis marmorata (which have been explained by bubble-induced vasospasms [4]) seen as heralds of more serious DCS manifestations [6].

### DCS type II

DCS type II comprises pulmonary manifestations as well as neurological manifestations.

DCS type II with pulmonary manifestation (also called 'chokes') is characterized by the occurrence of one or more symptoms of the classical triad of substernal pain, cough and dyspnoea occurring up to 12 hours after the dive and which may persist up to 48 hours. Contingent upon the extent of venous gas emboli load of the lungs, the condition may either dissolve over time or progress to loss of consciousness and ultimately death due to cardiac arrest or right heart failure [6].

DCS type II with neurological manifestations includes a broad variety of symptoms evoked by spinal cord or cerebral affection.

Spinal cord affection has been attributed to epidural vertebral venous system obstruction caused by gas bubbles [9]. This is potentially aggravated by stasis due to an increase in central venous pressure caused by venous gas emboli entering the lungs allowing for the coalescence of smaller bubbles. Typical symptoms include numbness, hypaesthesia, paraesthesia, weakness and paralysis of an extremity [5].

Symptoms of cerebral affection are mainly scotomas, visual field deficits, headache and dysexecutive syndromes. Cerebral affection has been attributed to arterialization of venous gas emboli (e.g. via a patent foramen ovale or other physiological shunts).

### Therapeutic strategies in DCS

The therapy strategy is mainly defined by the therapeutic options available. The gold standard of DCS therapy is recompression in a recompression chamber with subsequent slow decompression back to atmospheric pressure. During the initial stage and transportation to the chamber, oxygen breathing promotes bubble resolution and gas washout [6]. To balance diving-related dehydration, fluid replacement with i.v.-infusion of plasma expanders is recommended. Transportation via helicopter or aeroplane should be carried out at the lowest possible height [5].

## Mortality, autopsy results and management of barotrauma and DCS

The forensic medical examination, autopsy and assessment of diving-related fatalities require profound knowledge of diving physics, physiology and medicine and should be conducted by an experienced team. In the best-case scenario, autopsy is performed by a medical examiner experienced in the examination of diving-related fatalities accompanied by a clinically experienced diving medicine expert provided with relevant information by a police investigation unit experienced in the examination of diving fatalities. Experience of the team is all the more important as mistakes and omissions during equipment recovery may prevent the success subsequent procedures.

In this regard, a thorough examination should include documentation of the recovery site (including environmental conditions), the process of recovery and the body after recovery. Furthermore, water samples must be taken to allow for biological and chemical analysis. In practice, best information can be expected when police divers are involved in the taking of evidence, such as the examination and assessment of diving gear. These specialized units may also assist in the examination of gear such as diving computers. Meticulous inquiry of diving companions and operators, which is usually conducted by the police, may be essential for successful delineation and interpretation of medical conditions seen at autopsy.

Autopsy technique has to be adapted to the special situation of diving-related medical conditions. After initial inspection accompanied by photographic documentation, the apparel is removed. At this stage, radiological examination (usually postmortem computed tomography (PMCT)) is advised in order to obtain a full documentation of the deceased's body in a pristine state, to detect gas volumes in abnormal spaces, and to concomitantly assess medical conditions either pre-existing or due to the diving incident.

Interpretation of gas volumes detected during PMCT can be challenging since gas volumes in abnormal positions may be attributed to numerous mechanisms. In putrefied bodies, gas may be found in all compartments, impeding correct assessment. Gas analysis via airtight removal may prove the presence of putrescent gas or reveal a composition used for diving. Resuscitation efforts may artificially introduce gas in small volumes [16]. In addition, drowning is often accompanied by ingestion of air and water into the gastrointestinal tract, leading to triphase stomach content as depicted by Wydler's sign during autopsy. In regard to diving-related mechanisms, gas volumes may be detected based either on postmortem decompression artifact, DCS, or on pulmonary barotrauma. Whereas postmortem decompression artifact is a result of passive off-gassing due to decompression and results in gas bubble formation in various compartments (showing no discernible tissue reaction upon histological examination), DCS-related gas formation is pronounced in heart and blood vessels as well as lipid-rich structures. In pulmonary barotrauma, gas volumina result from lung rupture and are thus found mainly in the arterial and venous system. In the ascending diver, this will result in predominant brain affection. Other affected tissues include the pulmonary interstitium and subsequently the mediastinum and cervical tissues [6].

In addition, interstitial oedema of the lungs may be seen alongside frothy fluid content of the trachea. In airways and paranasal sinuses, a mixture of fluid and radio-opaque material (sand) may be noticed.

External examination is then continued. Besides common signs of postmortem changes and decay, it may reveal water-related stigmata such as washerwoman's skin of palms and soles as well as diving-related stigmata such as mask, face and suit squeeze, haemorrhage of the conjunctivae, haemorrhage or rupture of the tympanic membrane and haemorrhage of the middle ear. Emphysema of the cervical region may be seen as a result of pulmonary barotrauma of ascent [6].

Internal examination has to be adapted to the expected underlying pathomechanism derived from clinical, police and PMCT findings. If PMCT is not performed, autopsy technique has to be adjusted to detection of air embolism. Thus, craniotomy and cervical incisions should best be performed under water. For better delineation and photographic documentation of bubbles, the brain may be removed after clamping the cervical vessels. Alternatively, cervical vessels may be clamped and craniotomy performed with the head positioned above the body level. Formal neuropathological examination is advised after sufficient fixation in formalin. In some cases, perivascular haemorrhage of the brainstem may be noted. Spinal cord examination may show haemorrhages as well as infarcted tissue.

For assessment of gas trapped in the pleural cavities, PMCT yields best results. If PMCT is not performed, the pneumothorax probe is used to demonstrate gas in the pleural cavities. A skin incision in midsection is followed by undercutting fatty tissue and muscles. The resulting recess is then filled with water and incision of the intercostal muscles and the pleura cavity lead to discharge of entrapped gas. Alternatively, gas volumes may be collected using an airtight syringe inserted through the intercostal muscles and pleura.

In cases of suspected pulmonary barotrauma, lungs may be submerged and inflated with air to detect rupture sites. In addition to normal examination with regard to possible causes of acute coronary failure, heart dissection includes inspection of the foramen ovale to detect a patent foramen as a possible means of paradoxical air embolism.

In cases of diving-related fatalities a toxicological examination of samples collected during autopsy must be performed to rule out or confirm interference of relevant substances of abuse, such as alcohol and drugs, or prescription drugs. Blood may be tested for carbon monoxide, and collected gas volumes should be analysed to rule out or confirm a possible role of contamination of inhaled gas while diving and to determine the role of putrefaction in gas formation.

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# 32 Excited Delirium

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## Excited delirium described

An ongoing debate among medical specialties addresses whether the syndrome of excited (or agitated) delirium (ExD) exists and, if so, how to define it [9,10,14,22,25,38,40,41]. Our current understanding is that ExD or excited delirium syndrome (ExDS) is one of several terms that describe a syndrome, which has been employed among forensic investigators for decades and only recently by organized emergency medicine physicians. The DiMaios, forensic specialists, offer the following: ExDS involves the sudden death of an individual, in association with an episode of ExD, in which an autopsy fails to reveal evidence of sufficient trauma or natural disease to explain the death [5]. In 2009, the American College of Emergency Physicians issued a white paper on the ExDS [1], and the National Institute of Justice convened a workshop panel to examine the issue [17]. These are the two medical disciplines that typically encounter these patients, both clinically and postmortem.

ExD, a life-threatening condition, encompasses a syndrome that is broadly characterized by agitation, excitability, paranoia, aggression, great strength, and unresponsiveness to pain. It may be caused by several underlying conditions and is frequently associated with combativeness and elevated body temperature (Table 32.1) [43,45]. The common presentation of ExDS is shown in Table 32.2. Presently, the syndrome is largely associated with both drug intoxications and psychiatric illnesses [5,28,42]. Sympathomimetic agents, including cocaine, methamphetamine, epinephrine (adrenaline) and dopamine, predominate. A smaller cohort of ExD-affected people with sympathomimetic poisoning experiences malignant hyperthermia, which carries a risk of death regardless of police actions or method of subdual [37].

There are other forms of combative, agitated behaviour that require subdual. Such groups are often combined under the umbrella of emotionally disturbed people [35]. Emotionally disturbed people may be mistaken for people with ExD, and a subset of these may in fact display features of ExD. However, not all emotionally disturbed people who require subdual have ExDS. Although the term 'excited delirium' is of recent coinage, such conditions among mentally disturbed institutionalized patients, well before any type of medication for treatment, were thoroughly described by Bell ('Bell's mania') in the literature over 150 years ago [2].

ExD is a special type of delirium involving violent behaviour, as delirium and ExD are discrete entities [5,8,12,13,18,29,31,33–37,42–44]. Delirium in general is an acute, confusional syndrome with a transient disturbance in consciousness and cognition that has a variety of causes. Fatal instances are characterized by sudden death during or following an episode of ExD in which an autopsy fails to detect a disease or physical injury of a vulnerable site or degree sufficient to explain the death, and the circumstances are consistent with the syndrome [5]. It is a clinicopathological diagnosis based upon the autopsy and toxicological results evaluated in the context of the history and circumstances [9]. Table 32.3 presents the differential diagnosis of ExD.

ExDS-associated agitated behaviour often leads to law enforcement (LE) intervention and use of a variety of means of restraint or subdual within the accepted use-of-force spectrum, including neck holds, hog tying, pepper spray and controlled electronic devices (CED, commonly, Taser®) (Table 32.4) [11]. Typically, the victim continues to thrash about and struggle after being restrained [46–51]. An abrupt discontinuation of struggle, interpreted as 'playing possum', typically precedes an acute loss of vital signs. Medical responders at the scene characteristically confront a lifeless person with no cardiac activity (asystole or pulseless electrical activity (PEA) on ECG monitors) or bradycardia. Attempts at resuscitation are nearly always unsuccessful even if emergency services or physicians are at the scene.

The most common cause of PEA (a state where electrical activity can be recorded from the heart but there is not enough blood flow out of the heart to maintain a pulse or blood pressure), which is commonly the earliest recorded cardiac arrhythmia by emergency responders, is respiratory insufficiency leading to respiratory failure and hypoxia. Intravascular volume loss leading to hypovolaemia may exhaust cardiovascular compensatory mechanisms and result in PEA. Autopsy findings and laboratory results in emotionally disturbed people indicating dehydration and low haematocrit probably represent some degree of hypovolaemia. Hypoxia can diminish cardiac contractility by causing disturbances of inotropic substances, including

**Table 32.1 Characteristics of excited delirium**

| |
|---|
| Acute psychotic behaviour |
| Violent agitation |
| Altered mental status and delirium |
| Bizarre behaviours (e.g. jumping through window) |
| Profuse sweating |
| Incoherent speech (screaming and shouting) |
| Extraordinary strength and endurance |
| Lack of response to painful stimuli |
| Extreme exertion and hyperactivity |
| Hyperthermia |

**Table 32.2 Common presentation of excited delirium**

| |
|---|
| Acute onset of symptoms (minutes to hours) |
| Delirium |
| Combative and/or violent behaviour |
| Use of physical restraint and/or other forms of subdual |
| Sudden cardiac death [3] |
| Lack of response to CPR |
| History of either stimulant abuse or endogenous mental disease |

calcium and calcium–troponin interactions, and reducing intracellular energy reserves [24].

The predominant theory of the underlying etiology of ExD is an excess of catecholamines (such as adrenaline) or sympathetic nerve stimulation during the excited period. A syndrome, by definition, is a collection of signs and symptoms, not a specific disease. It is necessary for investigators to recognize that people with multiple conditions (see above) may present in this manner, including drug-induced psychosis, serotonin syndrome, diabetic ketoacidosis, paranoid schizophrenia, and others, such as alcohol withdrawal and head trauma. Recent research suggests that individuals with a history of chronic illicit stimulant abuse may be particularly susceptible to ExD.

It has been proposed by some observers that victims die of either respiratory arrest or fatal cardiac dysrhythmia. Diagnoses are ‘supported’ by postmortem examinations showing pulmonary and cerebral oedema with non-lethal self-inflicted injuries. In some cases of delayed death occurring in hospital, the victims often succumb to disseminated intravascular coagulation, rhabdomyolysis and renal failure. These fatal cardiopulmonary changes are thought to be the result of increased catecholamine stress on the heart, myocardial hypertrophy, microangiopathy and fatal arrhythmias. The cause of such pathophysiology may be unclear [38].

**Table 32.3 Differential diagnosis of excited delirium**

| |
|---|
| Substance intoxication |
| Substance withdrawal |
| Hypoxia |
| Electrolyte disturbances |
| Thyroid storm |
| Infection |
| Seizures |
| Head injury |
| Heat stroke |
| Serotonin syndrome |
| Neuroleptic malignant syndrome |

**Table 32.4 Excited delirium-associated deaths**

| |
|---|
| ExD-associated death after pepper spray |
| ExD-associated death after handcuffing/‘hog-tying’ |
| ExD-associated death after major physical struggle |
| ExD-associated death after TASER® use |
| ExD-associated death with no police |

In general, ExDS may have a mortality of about 10 per cent. Sympathomimetic agents include substances such as cocaine, methamphetamine, epinephrine and dopamine. There is a subset of ExD-affected people who have sympathomimetic poisoning with malignant hyperthermia sometimes associated with elevated serotonin levels [4]. These cases have a grim prognosis and are at high risk of death regardless of police actions or method of subdual. This correlates well with published observations that mortality is about 67 per cent for those with a temperature above 41.5°C (106.7°F).

The other cohort of ExDS cases and deaths is reported to be patients with psychiatric illnesses who abruptly stop taking their psychotherapeutic medications. Such forms of combative, agitated behaviour require subdual and are often grouped together under the umbrella of emotionally disturbed persons (EDPs). EDPs may be mistaken for people with ExDS, and a subset of these may in fact display features of ExD. However, not all EDPs that require subdual have the syndrome of ExD.

## Considerations in the investigation of potential ExDS-associated deaths

As most deaths occur during or shortly after subdual or attempted subdual by any combination of LE, bystanders or official institutional caregivers, investigators should be particularly meticulous in gathering, documenting and recording data with relevant materials. Table 32.5 highlights information that may be useful in establishing facts and should be considered during the death investigation. Assuming that the investigation and autopsy are performed and documented/reported in accordance with the National Institute of Justice’s *Death Investigation: A Guide for the Scene Investigator* [23] and the National Association of Medical Examiners’ *Forensic Autopsy Performance Standards* [27] (or the equivalent standards in other countries), additional information and procedures that may facilitate the determination of cause, manner and mechanism of death are displayed in Table 32.6.

**Table 32.5 Death investigation of excited delirium**

| | |
|---|---|
| 1 | A punctilious, frame-by-frame timeline of all events with attempts to verify, to the extent possible, the accuracy of the dates and times of reported events, with specific emphasis on the interval between subdual, unresponsiveness and death |
| 2 | Recent activities of the subject prior to the incident |
| 3 | The emotional state of the subject |
| 4 | The subject's reaction to each type of manoeuvre for subdual |
| 5 | The subject's medical conditions as determined by medical history, medical record review and medical conditions determined at autopsy |
| 6 | The subject's drug use history, including prescription and illicit drugs as well as alcohol |
| 7 | Specific inquiry into the subject's cardiac history, including review of any electrocardiograms or other cardiac function or laboratory tests, which have been performed in the past |
| 8 | Specific inquiry into the subject's seizure history to rule out history of seizures or to clarify the nature of a past seizure disorder |
| 9 | Review of witness accounts, police reports, use-of-force reports, emergency medical services records, medical and psychiatric records, and any videos, photographs or digital images of the events |
| 10 | Determination whether body temperature and ambient temperature were established and documentation of dates and times of such recordings |
| 11 | If death occurred after arrival at a hospital, obtaining blood drawn upon arrival at the hospital so it may be tested for intoxicants, including medications, if needed |
| 12 | Investigation of the subject's place of residence or last place to visit to determine if additional medical history or evidence of drug use exists |

## Considerations in the determination of cause and manner of death

In many cases, as noted, there are multiple forms of subdual or restraint such as neck holds (e.g. carotid sleeper hold; see 'Neck Holds' below), pepper spray, handcuffing, hobbling, 'hog-tying' slaps, asp baton strikes, chest compression, CED deployment and others. Because it is difficult to differentiate contributory methods from non-contributory ones, and because of limited space in the 'how injury occurred' section of the death certificate, it may be best to be generic in these complex cases and simply state that multiple forms of subdual or restraint were used. Of course, if there is reasonable evidence that one or more specific forms of subdual or restraint did cause death, such cases can be so certified. In general, in these cases, any form of subdual should be considered a stress of a magnitude that is comparable to other components of subdual.

**Table 32.6 Determination of cause, manner and mechanism of death in excited delirium**

| | |
|---|---|
| 1 | Performance of a complete autopsy of the scope usually performed for deaths in custody with appropriate histological sampling of organs and vitreous electrolytes |
| 2 | Comprehensive forensic toxicology of autopsy specimens and any retained antemortem samples [12], specifically including tests for:<br>• ethanol (intoxication/withdrawal)<br>• nervous system stimulants (sympathomimetic drugs, cocaine, amphetamines, caffeine)<br>• common drugs of abuse in the catchment area<br>• anti-seizure drugs<br>• valproic acid<br>• therapeutic drugs often prescribed for psychiatric disorders, antipsychotic agents (tricyclic antidepressants, monoamine oxidase inhibitors, methylphenidates, sedative drugs, sedative drug withdrawal, lithium, haloperidol)<br>• phencyclidine, benzoylecgonine, marijuana, lidocaine<br>• methylenedioxypyrovalerone, the most commonly identified active ingredient in 'bath salts' [26]<br>• hallucinogens<br>• lysergic acid diethylamide (LSD)<br>• mescaline<br>• phencyclidine (PCP)<br>• psilocybin<br>• anticholinergic drugs, includes drugs with anticholinergic side effects, antihistamine, corticosteroids<br>• over-the-counter drugs, e.g. dimenhydrinate and diphenhydramine |
| 3 | Determination of the nature of any other forms of subdual or restraint that were employed in the case in question |
| 4 | Removal and evaluation (interrogation) of any implanted cardiac or other electronic devices |
| 5 | Utilization of appropriate consultants such as cardiologists, pharmacologists, toxicologists, cardiac pathologists and neuropathologists, as needed |

## Should investigators collect and send fresh brain samples for anatomic and molecular evaluation in deaths suspected of ExDS?

Many LE and certainly spokespeople for Taser® urge medical examiners to collect such samples as soon as possible after an in-custody death. For example, a Taser® International representative sent an email to LE and the medical examiner after the death of a man hit by a CED:

> 'The attending medical examiners should urgently know the University of Miami Brain Endowment Fund is available with cutting edge research center that can determine drug abuse and look for excited delirium markers. This is one critical act that many ME's miss out on because of delays. It is imperative to get this info to the ME as the brain tissues must be collected ASAP.'

Their purpose is to establish that subdual by LE or use of a CED played no role in death.

In the research arena, anatomical and molecular evaluation of ExDS patients who die has focused primarily on postmortem brain examinations. Results from this increasingly robust body of work demonstrate a characteristic loss of the dopamine transporter in the striatum of chronic cocaine abusers who die, with clinical presentations consistent with and a diagnosis of ExDS. This suggests that one potential pathway for the development of ExDS is excessive dopamine stimulation in the striatum, but the significance of this in the larger context of ExDS unrelated to chronic cocaine abuse remains unknown. Heat shock proteins are measured directly in frozen brain tissue taken from Brodmann area 22. A detailed account of the method is contained in the 2009 paper by Mash et al. [20]. The problem with these biomarkers is that they can only be measured in fresh or rapidly frozen brain. The testing itself is difficult and costly. To sum up, there is currently no scientific basis to conclude that sending such fresh brain samples would assist the death investigator in determining whether ExDS occurred in a specific case [20,21].

## Neck holds

Neck holds, commonly and incorrectly called choke holds, in various applications may cause lethal asphyxia by that form of restraint, rarely by airway constriction or by carotid artery compression [30,39]. Some jurisdictions have outlawed such holds in most circumstances [32].

Anatomically, the neck is a complex structure. In the context of neck compression, the critical structures are the airway, arteries and veins, bones (hyoid and cervical spine), nerves (vagus, spinal cord), tongue and cartilages. The carotid bodies gauge $O_2$ and $CO_2$ in blood and, with hypoxia, trigger an increase in heart and respiratory rates. Carotid sinuses measure blood pressure; with hypotension, these receptors may increase cardiac output and cerebral blood flow via the autonomic nervous system. Compression of the carotid artery may cause stroke, and potentially death, particularly in the elderly, by interruption of adequate blood flow to the brain (in spite of the contribution from the vertebral arteries) or dislodgement of atherosclerotic plaques that embolize. Compression of one or both carotid sinuses may trigger either bradycardia or asystole. Vagus nerve stimulation slows the heartbeat. In view of these potentially life-threatening hazards, Dick [7] and emergency medical specialists recommend use of the jaw thrust airway manoeuvre, which hyperextends the neck by using the hands on the jaws and is incapable of causing airway occlusion or vagal stimulation.

Various types of neck hold have been used by police during attempted subdual, but most have fallen into desuetude because of the associated risks (Table 32.7). Because the risk of injury or death from the carotid sleeper is deemed by police to be less than the application of fists and batons, this form of restraint is considered appropriate for younger people. It requires 7734 $\text{kgf/m}^2$ of pressure to occlude the carotid arteries; if both are firmly compressed by a neck hold, loss of consciousness ensues in 10–15 seconds; 3094 $\text{kgf/m}^2$ of pressure occlude the jugular veins, effectively compromising outflow of blood from the brain and face [6]. The theory of carotid sinus stimulation inducing bradycardia and asystole to date has rested on a matter of exclusion [15,16], as there is no anatomical expression at autopsy to support this pathophysiology, and there are no reports of death [30]. Virtual absence of death related to carotid sinus syndrome – even in the more susceptible elderly – strongly militates against this mechanism in the younger population [19].

**Table 32.7 Common types of neck hold**

| Type of neck hold | Description |
|---|---|
| Mechanical hold | A device such as a baton is placed over the anterior neck. The restrainer uses both hands to pull backwards, and may injure the airway and compress the carotid arteries. |
| Bar hold (also known as the bar arm hold or choke hold) | This is similar to the mechanical hold, except that the 'device' is the restrainer's forearm. |
| Carotid sleeper hold (also called variously the sleeper hold, carotid restraint or later vascular neck restraint) | The forearm of the restrainer is flexed at the antecubital fossa over the anterior neck; designed to compress the carotid arteries, not the airway. |
| Shoulder pin restraint | This is a variation of the carotid sleeper in which one of the subject's arms is hyperextended at the shoulder and pinned against their neck; also designed to compress the carotids and spare the airway. |

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# 33 Suffocation/Asphyxiation in Childhood *Differential Diagnosis to SIDS*

*Jan-Peter Sperhake and Ann Sophie Schröder*

## Asphyxiation

In principal, those mechanisms leading to asphyxiation in infants and children are the same as in adults. Causes of asphyxiation include obstruction of arterial blood supply to the brain (e.g. in strangulation, choking), impaired venous blood return from the head (e.g. neck and/or chest compression, strong flexion of the neck, head-down position), or breathing impairment (e.g. chest compression, closure of the breathing orifices by covering or gagging, aspiration of foreign bodies). Besides those mechanically influenced causes, sometimes referred to as external asphyxiation (although the nomenclature is not unambiguous, see Chapter 21), there are non-mechanical or internal causes, which occur in the young (e.g. carbon monoxide intoxication, rebreathing of carbon dioxide, low-oxygen atmosphere, abnormalities of the larynx, infection with swelling of the epiglottis, etc.). Even though there are no fundamental pathophysiological differences between the various forms of asphyxiation between children and adults, children are more prone to the risk of asphyxiation due to their relative physical weakness and the underdeveloped competence in recognizing potential hazards and acting properly. By detecting injury patterns, reconstructing individual accidents, and identifying previously unrecognized dangers, the forensic pathologist can be an important player in preventive medicine [4,13].

Asphyxiation in childhood is a rare (nonetheless often preventable) event. In England and Wales in a 2-year-period, 136 children died of mechanical asphyxia, 65 per cent of whom were under 3 years of age [23]. The annual mean incidence of asphyxiation was 0.7 per 100 000 children under the age of 15 years, and 3.8 per 100 000 infants under 1 year. The sex ratio of girls to boys in this study was 1:2.7. In an Australian study the annual rate of accidental asphyxiation in children under the age of 15 due to upper airway interference such as facial occlusion, head and neck entrapment, rope or cord strangulation or foreign body aspiration was 0.47 per 100 000 children and 2.01 per 100 000 infants under 1 year of age [1]. The most frequent cause of death in this study was strangulation due to ropes and cords.

Establishing the correct diagnosis in cases of asphyxia in children is challenging and can even be impossible in some cases [7]. The forensic pathologist has mainly to rely on the findings of the death scene investigation in conjunction with the external and internal findings of the body. In most cases the original scene has already been altered by the caretakers or other persons who recovered the child, thus demanding a careful reconstruction. It must be taken into consideration that a reconstruction can be stressful and traumatizing for the caretakers and therefore must be undertaken with empathy and thoughtfulness. External findings on the body may be subtle but are nonetheless of great importance. Petechial haemorrhages, abrasions, bruises, skin reddening (sometimes patterned) and distribution of postmortem lividity can guide the forensic pathologist to the reconstruction of the posture and to the cause of the asphyxia. Petechiae make it possible not only to deduce venous congestion but, by their topographic distribution, also to reconstruct the level in which the congestion occurred. However, like most findings in asphyxiation cases, facial petechiae are not specific for asphyxiation as such. They occur in different circumstances, some of which are not associated with accidents or violence (e.g. excessive coughing or vomiting – Figure 33.1) and even in sudden infant death syndrome (SIDS) [11]. Since mechanisms involved in suffocation do not necessarily lead to venous congestion, the absence of petechiae does not exclude asphyxiation. Autopsy findings in cases of asphyxiation depend largely on the underlying mechanism. Internal findings such as petechial haemorrhages of the serous layers, fluid blood, dilation of the right heart, cyanosis and congestion are also non-specific [6] and therefore must be evaluated taking into account the external findings and circumstantial evidence.

Especially in cases of sudden and unexpected deaths of infants, the clear-cut differentiation between natural and non-natural death poses great difficulties for medicolegal experts. This is true in suspected accidental deaths as well as in homicide cases. In contrast to older children, who often display the same physical signs of violent asphyxiation as adults (e.g. laryngeal fractures, petechial haemorrhages, skin abrasions on the neck, defence injuries), such diagnostic hints are frequently missing in babies. One of the reasons is that it does not take a great physical effort to effectively close the breathing orifices of an infant, thus leading to no or minor external and internal injuries.

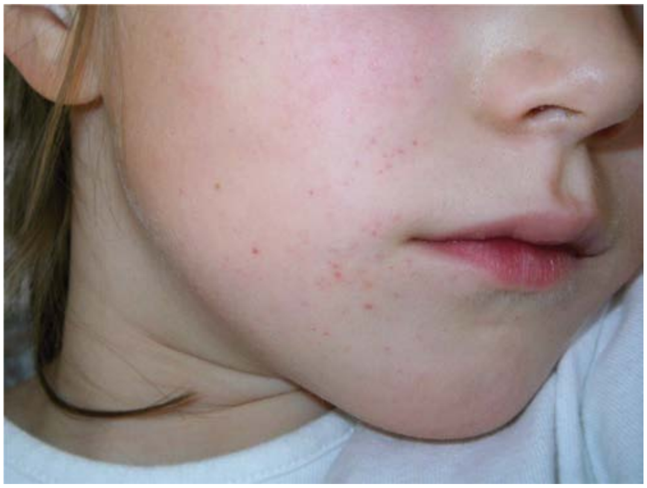

**Figure 33.1** Perioral petechiae in an 8-year-old girl after repetitive vomiting (observed by one of the authors on his daughter).

A baby cannot oppose the perpetrator and therefore defensive injuries are frequently absent. Finally, even macroscopic and microscopic autopsy findings are often of minor value due to their limited specificity. Inevitably, these diagnostic challenges lead to a certain dark field of unrecognized infanticides, even if all sudden deaths in infancy undergo a meticulous autopsy (which is not the case in many jurisdictions). Nonetheless, SIDS cannot be excluded with certainty in some cases of assumed asphyxiation, be it at the autopsy table or in court, when a clear-cut cause of death cannot be diagnosed. However, in a large German case-control study comprising 339 sudden deaths in infancy, 5 per cent of all cases initially thought to be SIDS turned out to be from unnatural causes, some of which were accidents [2].

The differentiation between accidental asphyxiation and SIDS is a difficult task. On the one hand, there are definite accidents such as strangulation by a loose cord or wedging of the infant between bed and wall; on the other hand, SIDS cases occur without any indication of a mechanically influencing factor. However, in forensic case work an overlap between accidents and SIDS certainly can be found. Some of the postulated pathomechanisms for SIDS, such as rebreathing of carbon dioxide and insufficient lung ventilation due to incomplete or complete closure of the airways by a soft mattress, include elements of an asphyxial death. There are also fatalities of small children that are clearly attributable to accidental strangulation or positional asphyxia, sometimes raising the question of legal responsibility and consumer safety when transportation devices or restraint systems are involved.

## Sudden infant death syndrome

The most comprehensive definition of SIDS has been given by Krous et al. [16]:

> ‘the unexpected death of an infant < 1 year of age, with onset of fatal episode apparently occurring during sleep, that remains unexplained after a thorough investigation, including performance of a complete autopsy and review of the circumstances of death and the clinical history.’

Dependent on the story, extent and results of the medicolegal investigation, documentation and other factors, five different categories are defined:

- *Category IA SIDS* – classic features of SIDS present and completely documented.
- *Category IB SIDS* – classic features of SIDS present but incompletely documented.
- *Category II SIDS* – classic features of SIDS with $\geq 1$ exception or additional feature respectively, including ‘mechanical asphyxia or suffocation caused by overlaying not determined with certainty’.
- *Unclassified sudden infant death (USID)* – deaths that do not meet the criteria for Category I or II SIDS but for which alternative diagnoses of natural or unnatural conditions are equivocal, including cases for which autopsies were not performed.
- *Postresuscitation cases* – ‘temporarily interrupted SIDS’.

Category II SIDS and USID pose the greatest challenges in distinguishing natural from non-natural deaths. However, there have been other proposals on how to implement asphyxiation in the classification of sudden unexpected deaths in infancy [26].

## SIDS vs accidental suffocation

‘Classic’ risk factors for SIDS such as cigarette smoking of the parents or sleeping in a prone position are less frequently distributed compared with the situation decades ago due to successful prevention campaigns. A side effect is an increasing proportion of cases with dangerous sleeping positions among the few SIDS cases occurring today. Obviously, any babies in potentially hazardous sleeping conditions remain at risk despite the success of prevention campaigns. Today, an integral part of the definition of SIDS is a thorough death scene investigation. The surroundings in which the infant is recovered often suggests a mechanical asphyxia. The proportion is estimated up to 50 per cent [15,20,24]. Asphyxia must be considered in sofa-sharing cases and SIDS occurring in the parental bed. In these cases, it is not always possible to reconstruct the original situation accurately, because the original scene has often been changed by the person recovering the baby. Often it remains unclear whether the mouth and nose were covered or closed by the body of a co-sleeping person, bed clothing or the back rest of the sofa. Wedging situations with impairment of the baby's breathing movements do occur in bed-sharing situations but are very hard to reconstruct with certainty. Arrangement and blanching of postmortem lividity can be helpful in cases with a postmortem interval of at least several hours before discovery of the dead baby, thus leaving enough time for the lividity to be fixed in adjacent body parts. According to many studies, the risk for SIDS in bed-sharing situations is higher in infants younger than 4 months, and especially high in very young babies [27,29]. Carpenter et al. [9] calculated a fivefold increased risk for SIDS in infants younger than 3 months who shared the bed with a parent, even if the infants were breastfed, both parents were non-smokers and the mother consumed neither alcohol nor drugs. Some sudden infant deaths in maternity wards might be explained by mechanisms leading to oppression of the tender newborn resulting in insufficient ventilation of the lungs [15]. However, dangerous sleeping situations also occur in older infants and apart from bed-sharing situations.

### Case 1

A 9-month-old male infant was found dead after sleeping alone in a double bed in prone position. The boy had been put on an extremely soft mattress made of foam material. Underneath the mattress, two slats of the bed were missing, thus creating a 'crater' in which the baby was sleeping (Figure 33.2). According to the parents, the boy was completely covered by a duvet weighing 3.5 kg when he was found. The death scene investigation was performed 2.5 hours later by a medicolegal expert. The infant's body and clothing as well as the surrounding bed linen were wet with sweat. Body temperature at that time was 37.8°C.

It remains unclear whether this case is a SIDS case in its classical meaning. Obviously, the boy was not capable of disengaging himself from a situation in which, on the one hand, massive overheating occurred and which, on the other hand, hampered breathing freely. Therefore, discrimination between a SIDS case and a 'mere' accidental death remains impossible. The case is accurately classified as USID.

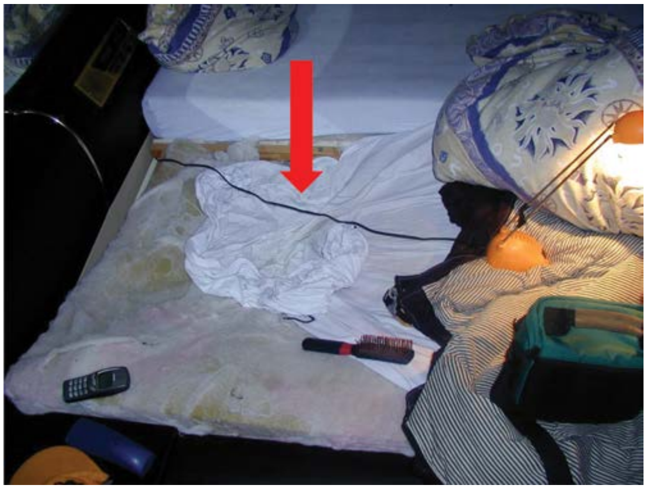

**Figure 33.2** Case 1: Bedding situation with a 'crater' resulting from the lack of slats in the frame.

## ■ Inflicted asphyxiation under soft covering

Inflicted asphyxia of an infant by closing the nose and mouth is often hard to diagnose or even undetectable by the external examination as well as by the autopsy. To effectively close the external airways, it is sufficient to press a pillow, plastic bag or hands with moderate weight on an infant's face. Killing a baby in this manner does not require brute force. That is why subcutaneous haematomas, scratches or abrasions in the perioral region, in the mouth or at the nose are often absent. Nonetheless, it is crucial to inspect the skin carefully for even minor injuries. Facial or conjunctival petechiae are not a common feature, because closing of the mouth and nose results in an obstruction of the air flow but not of the venous return of blood. The serous layers of the lungs and heart may show reddish spots (Tardieu spots), but this finding is inconsistent and not specific, and can also be found in SIDS cases.

In histology the lungs may present with hyperinflated areas, tears of the alveolar septae and haemorrhages. These findings are also not specific for inflicted asphyxia and might also occur in accidental closure of the airways or as a sequel of mechanical ventilation in resuscitation attempts. Intra-alveolar haemosiderin ('storage iron') has been interpreted as previously imposed suffocation (e.g. as in Munchausen syndrome by proxy [2,3]) or preceding so-called 'near-miss' episodes [28]. Today, the assumption that intra-alveolar haemosiderin is an indicator for imposed suffocation attempts has been relativized by more recent publications [18].

### Case 2

A 9-week-old baby boy allegedly was found dead by his mother in the morning hours in a supine position. Resuscitation attempts remained unsuccessful. The findings of the medico-legal autopsy were compatible with SIDS. No external injuries were present. The lungs were moderately hyperinflated. The visceral layer of the pleura showed scattered petechial haemorrhages. Histologically, minor intra-alveolar haemorrhages were present but no haemosiderin was found.

A couple of days after the autopsy the mother confessed to friends and relatives to having actively smothered her son by closing his mouth and nose with a blanket. She was arrested and charged with manslaughter. In court she withdrew her confession and attributed it to the exceptional psychological state that she was in after she had found her baby dead. The medicolegal expert witness was not able to differentiate between SIDS and inflicted asphyxia on the

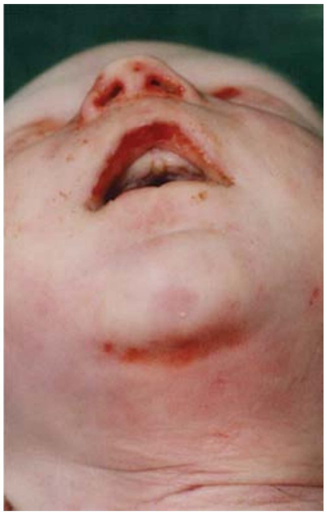

**Figure 33.3** Case 3: Facial abrasions suggesting covering of the mouth and nose.

basis of his autopsy findings. The mother was cleared of all allegations for want of evidence.

## Case 3

A 6-year old boy allegedly was found lifeless by his parents in the top of a pram in the early morning hours without any previous illness. At the scene, the room was rather warm (23.4°C) and smelled of cigarette smoke. The boy was covered with a blanket and had a pillow for his head.

At the external examination, dry skin abrasions were discovered at the base of the nose, nostrils, mouth and chin (Figure 33.3). In addition, haematomas at the mandible, behind the right auricle and in the right palm were obvious. The boy was malnourished.

At autopsy, 18 bone fractures of different ages, thymic involution, alveolar haemorrhages and a healing brain contusion of the right parietal lobe were the main findings. The facial subcutaneous tissue showed discrete haemorrhages underneath the abrasions. Confronted with these findings, the father confessed to having smothered his son by closing his mouth and nostrils using his hands.

## Positional asphyxia and wedging

Mobile infants, toddlers as well as older children are at risk of manoeuvring their bodies into positions which might result in impaired breathing, restriction of the venous return of blood from the head or narrowing of the respiratory passages. Also, combinations of those mechanisms can occur. Petechial haemorrhages in the face but also in the region of the pectoral girdle are a common feature. For the medicolegal reconstruction of the accident, the distribution and the pattern of postmortem lividity and pressure marks play an important role.

Wedging occurs in different situations, often involving modified cribs, mattresses and other sleeping devices [5,8] or insufficiently secured objects (e.g. closets) in the home environment.

## Case 4

A 16-month-old boy played around in a café while his mother had a cup of tea and chatted with a friend. Toys were arranged in a corner of the café. After a while, the mother found the boy lifeless in a fetal curl, lying in a wooden toy box measuring 30 cm in height, with a top opening of 40 × 28 cm (Figure 33.4a). Extensive resuscitation attempts remained unsuccessful.

At autopsy the most striking findings were petechiae in the eye lids (Figure 33.4b) and conjunctivae and small subcutaneous haematomas at the forehead. The child's body weight was 11.1 kg and his height was 79 cm.

Reconstruction of the events with a 12-month-old boy of similar stature (height 82 cm) in a flexible box with the dimensions of the toy box showed that a toddler can climb in the box on his own. The side position in the narrow box results in a fetal curl, pressing the chin on the chest and making it impossible to get out without help (Figure 33.4c).

Positional asphyxia due to a hindered venous return from the head was the most likely diagnosis.

## Case 5

An 8-month-old boy fell asleep after lunch in a pushchair without the safety belts fastened. Two hours later, he was found unresponsive and hanging out of the pushchair with his chin held in position by the bumper bar. Resuscitation attempts initially were successful. An immediate medicolegal examination was not requested. Brain death was diagnosed 4 days later. There were no indicative skin marks visible at the external examination. The scene was reconstructed with the permission of the parents and is shown in Figure 33.5.

At autopsy, no specific signs of asphyxiation such as petechial bleedings or marks on the neck were present.

## Case 6

A 26-month-old boy was found dead in an infant's travel bed. The boy has been described as being 'very wild'. For that reason, the mother had 'secured' the travel bed by putting a wooden cot upside-down on the top of it, thus creating a 'cage', in order to prevent her son from escaping (Figure 33.6a). By loosely fixing the rear part of the cot frame with textile material on the top of the travel bed, the mother created hinge-joints, allowing the 'cage' to be opened to one side (Figure 33.6b). When she found the boy dead, he was standing with his head caught between the half-opened top of the 'cage' and the semi-stiff side wall of

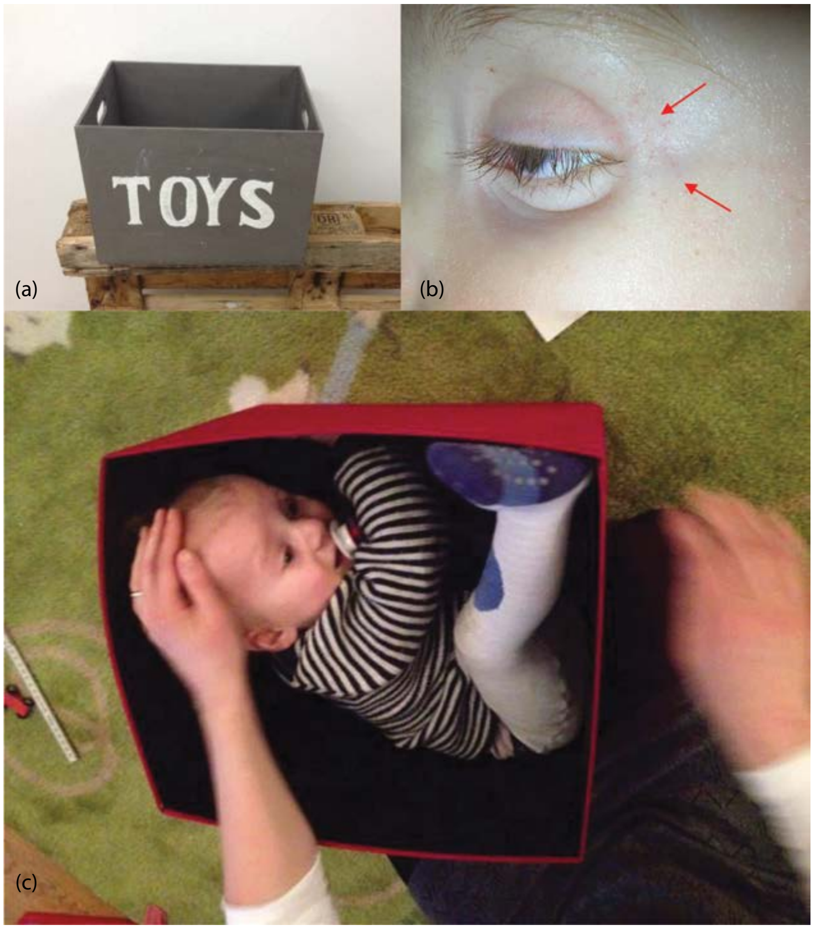

**Figure 33.4** Case 4: (a) Toy box; (b) facial petechiae; (c) reconstruction with a boy (son of one of the authors) and a box of the same dimensions.

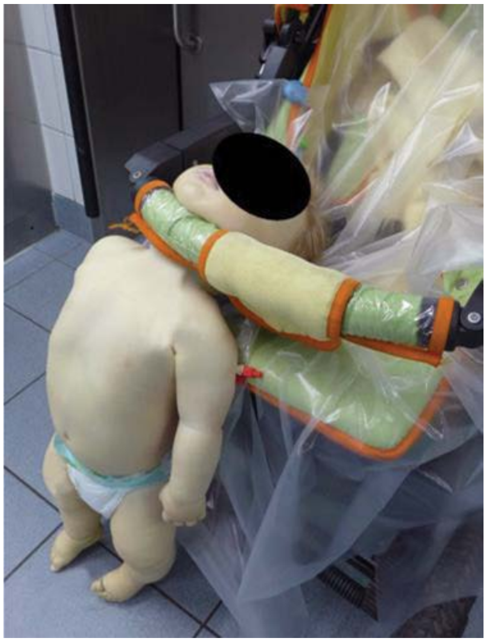

**Figure 33.5** Case 5: Position of the boy when he was found unresponsive (reconstruction with permission of the parents).

the travel bed. Apparently, he had tried to free himself and finally got trapped in this situation.

External examination of the body revealed streaky scratches on the right cheek and auricle, in the left lower neck, as well as on the forehead (Figure 33.6c,d). Petechial haemorrhages were present in the conjunctivae and eye lids. Apart from hyperinflated lungs, the internal findings were normal.

Death was attributed to asphyxiation due to strangulation of the neck.

## Case 7

A 10-year-old boy was playing unattended in the basement of a house. Later in the day he was found dead by his mother. The boy had been trapped by a pile of plasterboard (sheetrock). Nine sheets of plasterboard, measuring 125 cm in height and each weighing 10 kg, had been stored in the basement, leaning upright on the wall of a narrow corridor. The whole pile fell from the wall towards the opposite wall, where it got caught on the neck of the boy just underneath the chin.

External examination and autopsy revealed horizontal abrasion marks on the neck and the right lower arm, as well as abrasions and subcutaneous haemorrhages in the upper parts of the back. The larynx was intact. The facial skin was pale and without any petechiae.

Death was attributed to compression of the carotid arteries.

## Foreign body aspiration and suction-type suffocation

Infants and small toddlers are at high risk of aspirating any kinds of object that are small enough to fit into larynx and trachea [12,14]. Small, rounded food such as peanuts, raisins and pills, as well as small parts of toys, can be dangerous objects if they fit into the airways of the child. In those cases, a pre-autopsy postmortem computed tomography (PMCT) scan is helpful to visualize the foreign body and to localize its position in the respiratory tract. In the absence of the aspirated object, it can be very difficult to establish the diagnosis at autopsy.

Furthermore, some household devices can also lead to the accidental closure of mouth and nostrils. Nakamura et al. [22] described 17 incidents in which a semi-rigid, often hemispherical object, such as stacking toys or containers, cupped the face.

### Case 8

The parents assumed that their 18-month-old, previously healthy son had choked on a pretzel stick. On the arrival of the emergency doctor, the boy's oxygen saturation was 65 per cent but could initially be improved with an oxygen mask. He was taken to a nearby hospital in which intubation and tracheotomy efforts were unsuccessful due to a rigid resistance in the airway. The boy died in hospital.

In a PMCT a hemispherical foreign body could be visualized in the larynx (Figure 33.7a,b). The boy showed an intensive upper venous congestion and haematoma of the anterior part of the neck. At autopsy the epiglottis was found moderately swollen and compromised by the rather large palatine tonsils. The main autopsy finding was an almost complete occlusion of the larynx by a fragment of a hazelnut shell level with the vocal cords (Figure 33.7c,d).

Death was attributed to mechanical asphyxiation due to foreign body aspiration.

## Self-strangulation

Apart from a large proportion of homicidal cases [30], strangulation in infants and children can occur accidentally as well as by suicide. One might assume that suicides are more frequent in older children and adolescents. However, there might be a grey zone of cases that might be either suicides or accidents due to autoerotic behaviour.

An Austrian study found 171 cases of self-hanging in adolescents aged 14 years or younger (with the vast majority older than 9 years), of which 88 per cent were boys [10]. The

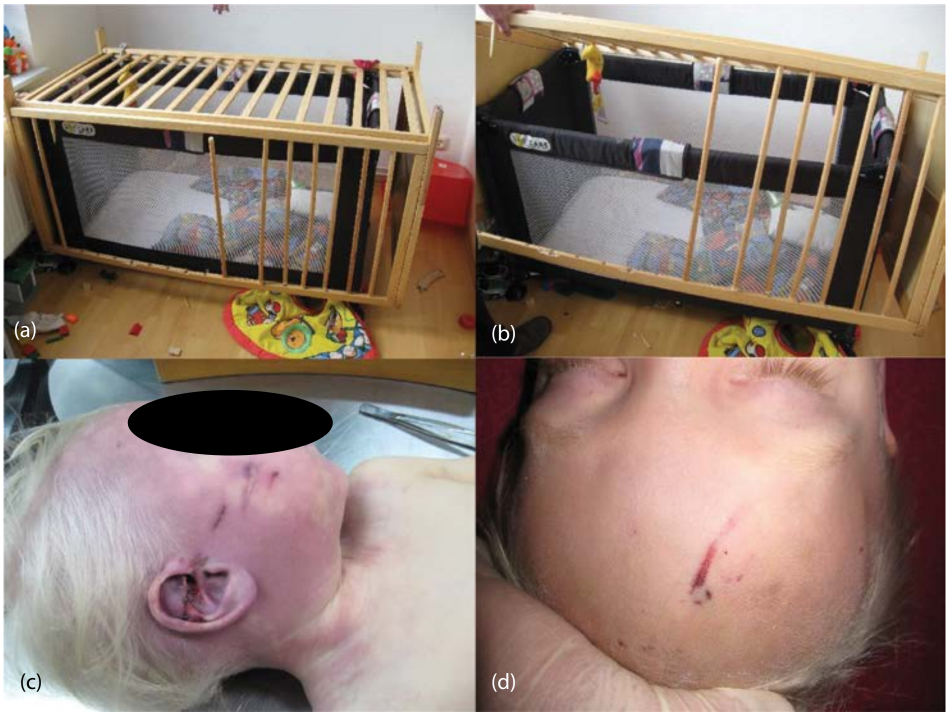

**Figure 33.6** Case 6: (a) Manually altered travel bed with parts of a wooden cot on top; (b) opening mechanism; (c) scratches and pressure marks on the check and (d) forehead.

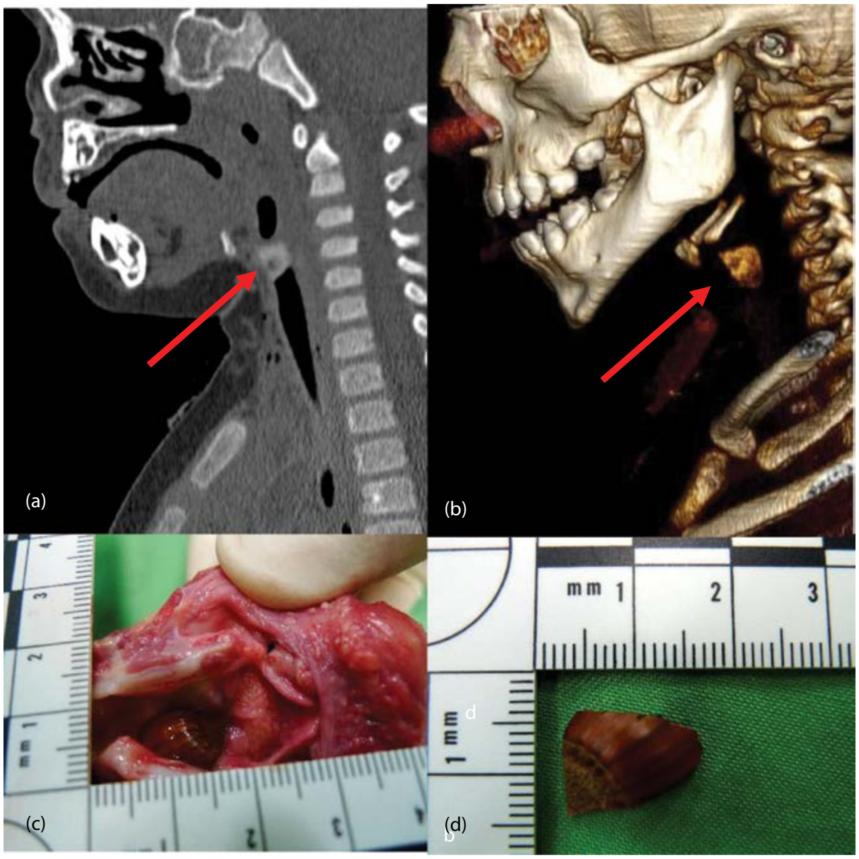

**Figure 33.7** Case 8: Aspirated hazelnut shell in the larynx with (a) 2D and (b) 3D reconstruction in postmortem CT. The hemispherical nut shell closed the larynx (c) and was 1.3 × 1.0 cm in size (d).

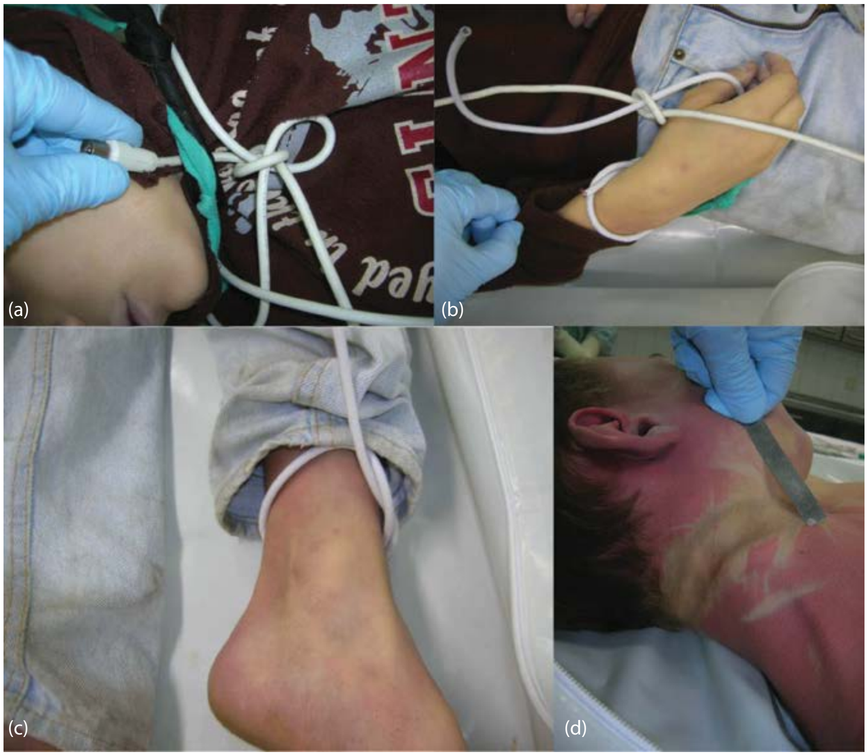

**Figure 33.8** Case 9: (a) Antenna cable and textile wraps firmly tied to the neck; (b),(c) bondage of wrists and ankles; (d) strangulation mark.

authors discuss a dark field of accidents but do not point to possible autoerotic accidents specifically. For example, in 2001, Le and Macnab [19] identified cloth towel dispensers as a potential hazard for children as part of a 'thrill-seeking' behaviour or choking games respectively.

In accidental cases, even strangulation of infants by human hair while co-sleeping with the mother are reported [21]. Children on playgrounds are at risk of being strangulated by drawstrings on clothing [25]. To avoid such accidents of self-strangulation it is recommended that clothing of infants and toddlers is not too wide and has no ribbons or drawstrings, and that small children do not wear any chains or toys attached to cords or dummy ribbons round their neck. String-like articles should be kept out of the reach of unsupervised playing or sleeping children. Children should always sleep in undamaged cots with appropriately normed distances between bars and/or grids. Infants and small children should not be left unattended in baby swings or baby buggies and it should always be ensured that the tethering straps are short and firmly applied and that there is no one-sided fastening.

## Case 9

A 13-year-old boy was found dead by his mother in a store room. His body was suspended in an upward position from a hook on the door with his feet in contact with the ground. An antenna cable and a textile strap had been firmly tied around the boy's neck (Figure 33.8a) and connected to the hook. His wrists and ankles had been bonded by the same cable (Figure 33.8b,c). The zip of his trousers had been opened. The boy had been diagnosed with attention deficit hyperactivity disorder and sometimes displayed inappropriate sexualized behaviour, such as stroking his mother's breasts and masturbating in front of his mother.

At autopsy there was a clear-cut ligature mark around the whole circumference of the neck (Figure 33.8d). Only a few petechial bleedings in the conjunctive and haemorrhages at the origins of the sternocleidomastoid muscles as well as in the frontal parts of the intervertebral discs were present as a sign of vital hanging.

It could not be clarified fully whether the boy died from suicidal hanging or from an autoerotic accident.

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# 34 Masking of Homicide

*Stefan Pollak and Annette Thierauf-Emberger*

## Introduction

Intentional simulation and dissimulation of criminal acts are behaviour patterns frequently met in both police investigations and forensic medicine. So, it is a well-known fact that some persons inflict injuries on themselves and then falsely claim to have been harmed by an offender. This also applies to alleged assaults to the neck, for instance in the context of feigned rape or robbery. Findings on the cervical skin may be reddening, scratches and even haematomas caused by self-pinching in order to pretend a fictional offence [12].

Even more worrying are homicide cases disguised as suicides, accidents or natural deaths, which may be misinterpreted due to secondary cover-up activities of the perpetrator. In addition to that, it is common knowledge that the postmortem examination of strangled victims is prone to errors [32]. The medicolegal and criminal literature contains an abundance of misdiagnoses attributable to the absence, insignificance or poor specificity of external findings [38]. Moreover, it has to be admitted that inexperienced, uncritical or unsuspecting investigators are at risk of overlooking minor signs of neck compression. A considerable number of homicidal strangulations are elucidated only belatedly, sometimes as a result of an unexpected confession, rumours and/or suspicious facts emerging at a later time.

A mere external examination of the deceased necessarily leaves part of the strangulation deaths undetected. Even hanging cases that should be readily identifiable in the opinion of most lay persons are not always diagnosed correctly. Typical reasons for erroneous assumptions include lack of knowledge about the possible manifestations of hanging, hasty judgement in seemingly clear situations, changes/manipulations at the scene and/or on the body as well as insufficient inspection of the deceased without adequate focus on the neck [32].

The topic of this chapter – masking of homicide by strangulation – is so diverse and extensive that a restriction of content seems necessary. Otherwise, one would have to cover all the activities of perpetrators intended to prevent the recognition and solution of the crime committed (e.g. concealment or disposal of the body, removal of the ligature or hanging device, arson in order to make the victim's identification and the investigation of death impossible). Even staging of an accident due to sexual asphyxia has been observed [31]. A thorough discussion of the all of these scenarios is beyond the scope of this chapter. The discussion here will concentrate on homicides disguised as suicidal hanging.

## Historical aspects

When going through medicolegal literature, one is staggered by the widely differing statements about the incidence of homicidal hangings. The famous British textbook author Cyril John Polson [39] still claimed in the 1960s that:

> 'there is … no report of the occurrence in this country of homicidal hanging, nor of suspension of the victim of murder, to simulate hanging.'

In contrast, in continental Europe a great number of relevant observations were published as early as the 19th century. Probably the most spectacular case is named after the strangled victim Toussaint-Augustin Gouffé, a 49-year-old Parisian civil servant. On 26 July 1889 he was killed by Michel Eyrand and his accomplice Gabrielle Bompard who wound the cord of a dressing gown around Gouffé's neck while pretending to flirt with him. Her partner in crime had hidden behind a curtain at the rear of Gouffé who was sitting in a deckchair. Eyrand caught the cord and hanged the unsuspecting victim using a pulley-like mechanism [19]. The elucidation of this affair is still regarded as a milestone in forensic pathology.

In the 11th edition of Hofmann's classic textbook [16] published in 1927, homicidal hanging is dealt with on three pages. The topic is treated using examples both from the Vienna autopsy material and from the subject literature. Hofmann already differentiated between homicidal hanging in the proper sense and secondary suspension of a person previously killed by some other method. In the first-mentioned category, Hofmann details a homicide-suicide in the course of which a man killed his five children by hanging before he hanged himself. Regarding the latter category, the author stated:

> 'It is not rare that victims killed in a different way are hanged in order to make the death appear as a suicide.'

In 1942, Maync [27] presented a review based on articles in German-language journals. He compiled 17 cases of homicidal hanging and 11 killings with subsequent hanging intended to simulate suicide. In his evaluation, Maync took the view that 'typical' hanging scenarios (characterized by free suspension of the body and a noose symmetrically ascending towards the occipital region) are more common in homicides than in suicides. From a present-day perspective, however, this opinion cannot be shared without reservation [35,36,41].

At present, reports on homicides presenting themselves as hangings are so numerous that a complete listing is not possible in this chapter. Nevertheless, it has to be emphasized that the occurrence of such cases is a global phenomenon and certainly not restricted to Central Europe [9,10,22,43,45,47,48,51,52,54].

## Epidemiology

In many countries, including Germany, hanging is the most common method of suicide. Therefore, death by hanging is often exclusively associated with suicide thus ignoring the realistic alternatives of accidental death or homicide [15,50]. According to Hofmann [16] the mere predominance of self-hangings may support the premature assumption of suicide, especially in examiners who have only limited experience. Authors who survey ample autopsy material suppose a high dark figure of overlooked crimes [41]. This problem was addressed by Böhmer in 1940 [6]:

> 'The risk of insufficient care is probably never as great as when examining hanged victims, which involves the possibility that a criminal act may go undetected.'

DiMaio [11] reviewed 133 homicides due to asphyxia comprising mainly ligature and manual strangulation (48 and 41 deaths, respectively); his study material included only one case of homicidal hanging. Russo et al. [42] recorded one homicide among 260 hanging deaths examined in a region of Northern Italy. Püschel et al. [41] reported six homicidal hangings from the city of Hamburg and its surroundings. In comparison with the total number of fatal hangings in the observation period, the authors estimated that the ratio of homicidal versus suicidal hangings is about 1:1000. In 2014, Sauvageau found a remarkably high rate of homicidal hangings in the Canadian province of Quebec: In a total of 251 hanging cases from a 6-year period, there were 8 accidents and 4 homicides, the latter constituting a proportion of 1.6 per cent [45].

## Simulation and dissimulation

In medicolegal practice, the examiner is often confronted with attempts to deceive. The motives for covering up the true facts are manifold [15]. In clinical forensic medicine, the spectrum of deception ranges from insurance fraud to falsely pretended assaults. In hanging deaths, suicides are frequently disguised by family members who remove the ligature and may even apply a chin strap to conceal the hanging mark. In cases of sexual asphyxia, evidence pointing to autoerotic activity may be 'cleared' so that the examiner at the scene will not think of an unnatural death. Self-suffocation caused by a plastic bag pulled over the head can easily be dissimulated by disposing of the suicide device. On the other hand, a homicide by smothering may be covered up as a plastic bag suicide [46]. From the criminalistic point of view, the dissimulation of a homicide by feigning a hanging suicide is especially challenging.

## Hanging as a way of killing

Some authors hold the opinion that homicidal hanging in the proper sense occurs only under certain conditions [16]. These include a considerable difference in bodily power between the perpetrator and the victim, the collaboration of two or more offenders [1,41,43], an impaired or lack of capacity to act on the part of the victim (e.g. due to previously inflicted injuries, mental retardation or physical handicap, drug and/or alcohol intoxication). Knight's textbook on forensic medicine stated [44]:

> 'For one individual to hang another, there must be either a disparity in their size and strength – or the victim must be drugged, drunk or otherwise incapacitated by fear, illness or senility.'

Undoubtedly, any impairment of the victim and physical superiority of the perpetrator are factors which facilitate homicidal hanging. Homicide-suicides involving children can be cited as an example [3,16,22,35,41,45]. Defenselessness of the victim may be caused by disturbed or lost consciousness due to alcoholization [41,43,53], drug or pesticide intoxication [41,48], blunt head trauma [25,41] or previous strangling [24,41]. On the other hand, several authors emphasized that a disparity in strength is not an indispensable precondition for homicidal hanging. The literature survey of Maync [27] mentions 6 out of 13 cases in which adult victims were hanged by single perpetrators without significant superiority of physical strength. Thorough planning, in combination with an element of surprise, may be just as effective as mere physical overpowering, especially in unsuspecting victims [40,41].

## Hanging of a homicide victim to simulate suicide

This group of offences comprises cases of hanging in persons who were severely injured or killed by a different method, such as blunt force, suffocation or manual/ligature strangulation, before the hanging (Figures 34.1–34.6). Therefore, the hanging act was not intended to cause the victim's death, but to make the fatality appear to be a suicide.

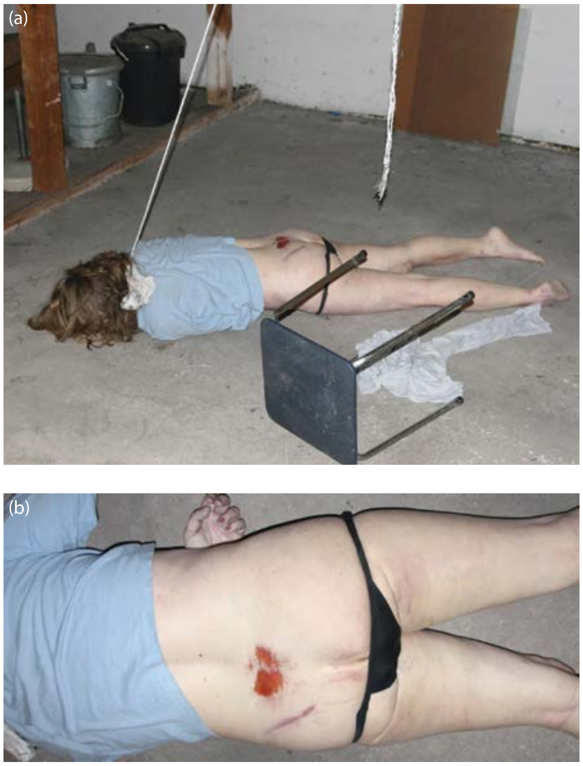

**Figure 34.1** (a), (b) Manual strangulation of a 47-year-old woman with attempted masking as suicide by subsequent suspension of the victim [36]. The body was found face-down in the attic where it had been taken by the perpetrator. The hanging device went up to the collar beam, but was not fixed to it. Note the incomplete clothing with the underpants pushed down and the waistband rolled in. The parched graze abrasion in the sacral region was caused by dragging the victim from the site of the crime (bedroom) to the attic.

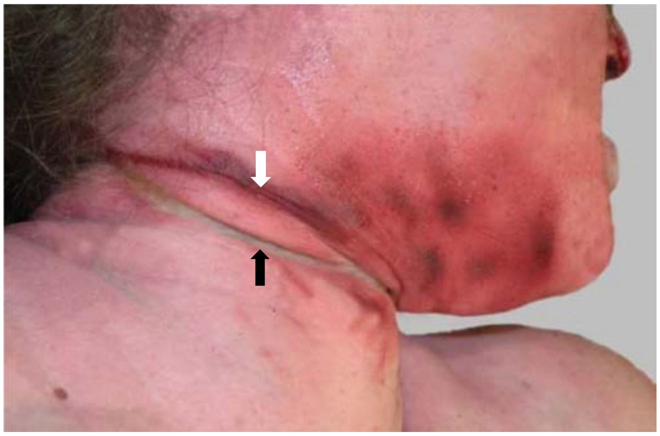

**Figure 34.2** Right side of the neck showing two hanging marks (arrows) and a multitude of haematomas from previous manual strangulation (same case as depicted in Figure 34.1).

- A crime may be suspected for different reasons. Examples include *conspicuous circumstances* such as rumours, allegations, a lacking motive of suicide or serious conflicts in the background as well as connection data of a suspect's mobile phone indicating that it had been logged into GSM radio cells covering the victim's domicile [36].
- In some cases, peculiarities at the *scene* can be indicative of homicide. Examples include the absence of a climbing aid (if necessary to reach the point of suspension), drag marks on the floor, blood soiling, more than one groove on a wooden beam from repeated attempts to pull up the body [17,20,36] or multiple colour abrasions on an object used for fastening the hanging device [41], too short a distance between the knot and the point of suspension (not sufficient to put the head into the noose before it tightens) [40,41], the presence of a special knot (typical of a profession not practised by the victim) [29].

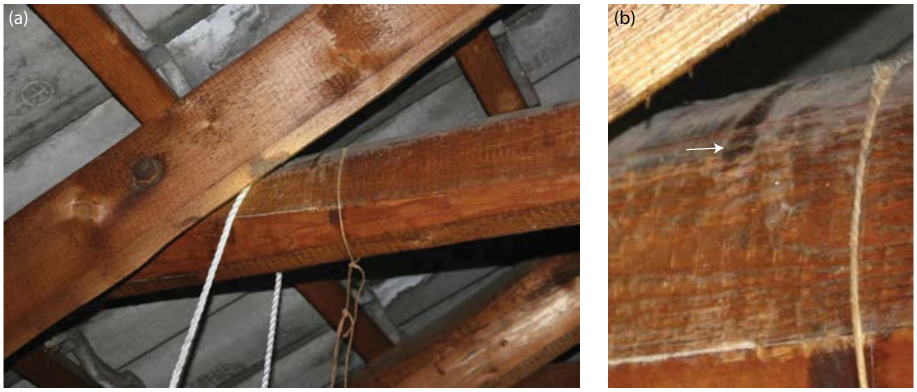

**Figure 34.3** (a) Wooden collar beam used for suspending the already lifeless victim manually strangled before (same case as depicted in Figures 34.1 and 34.2). The hanging device was a white, triple-twisted polypropylene rope running across the beam's upper surface without being fixed to it. (b) Note the additional traction mark (→) close to the final position of the rope (formed in a previous attempt to haul the victim up).

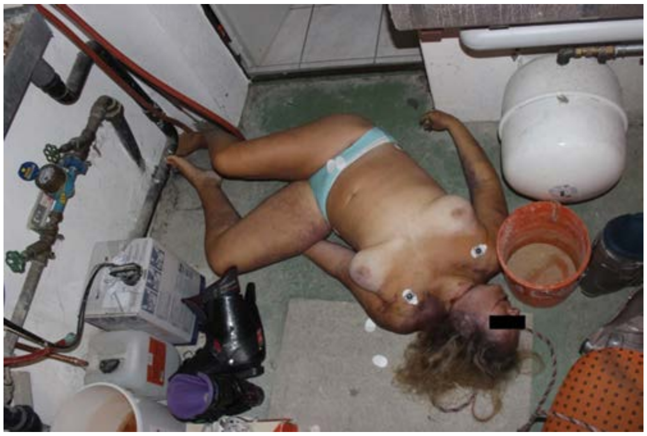
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**Figure 34.4** Manual strangulation of a 43-year-old woman who was subsequently hanged by the perpetrator to stage a suicide [13]. The body was found in the boiler room of her house, but the homicide had been committed in the bedroom. By the time the police arrived, the noose had already been removed by the first aider. Originally, the hanging device (plastic rope) was attached to a heating pipe; the victim's head was elevated about 10 cm above the ground. The woman was only wearing panties and her breasts were bare.

- The *outward appearance* and/or the *clothing* of the person found hanged may be striking. Examples of this are a naked or topless female victim [13,51], underpants pushed down and waistband rolled in [36], improper or incomplete clothing [17,41], garments put on back-to-front [21].
- From the medical view, *injuries* not attributable to suicidal hanging play an essential role when determining the manner of death. The following findings may be helpful to indicate suspicion:
    - Fingertip bruises, scratch-like and crescent-shaped abrasions on the neck skin [20,23,24,28,35,36,41], more than one hanging mark in the presence of a noose having only one ligature turn [36,51], a horizontal strangulation mark on the neck not being in line with the noose running upward to the point of suspension [41].

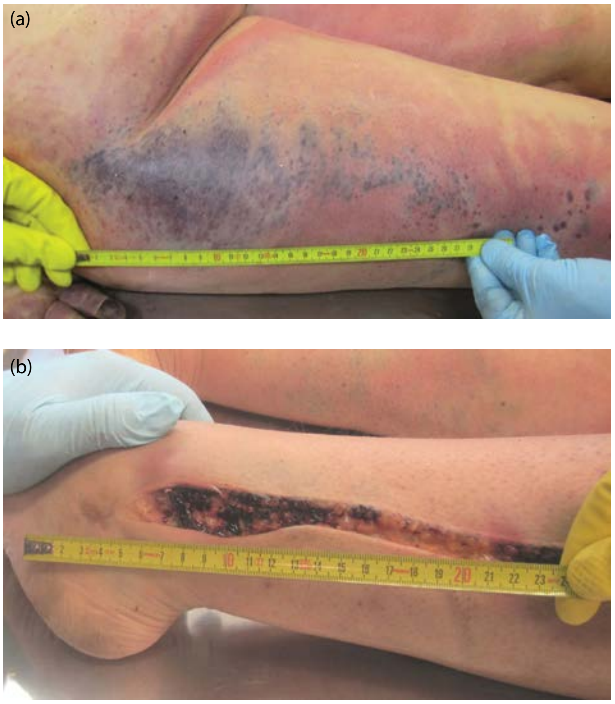

**Figure 34.6** (a), (b) Left thigh and lower leg with extensive contusions on the lateral aspects of the limb (same case as depicted in Figures 34.4 and 34.5). Dissection revealed blood extravasation in the subcutaneous layer and the muscle tissue, probably caused by dragging the overweight woman over the stairs. The haematomas can be regarded as a sign of vitality indicating that the heart was still beating when the victim was taken to the site of the hanging.

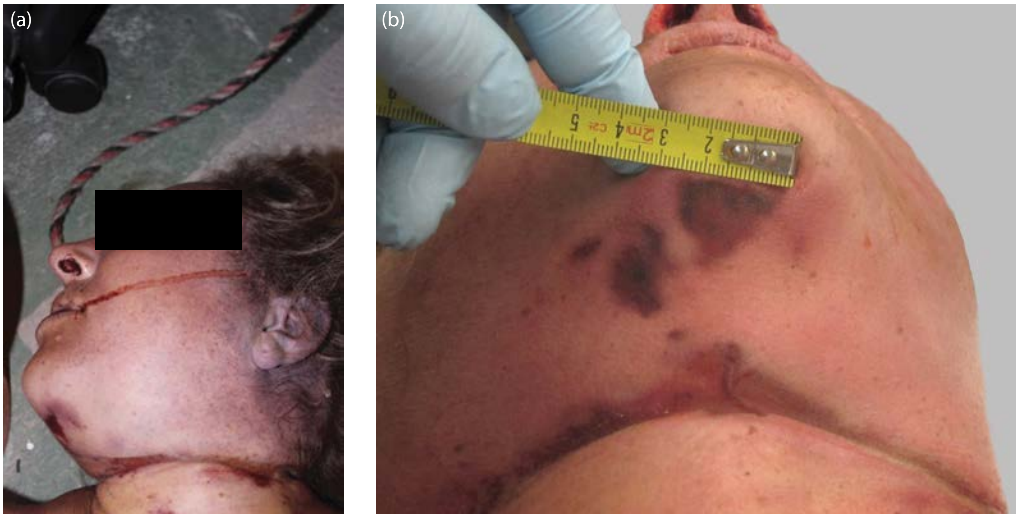
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**Figure 34.5** (a) Left side of the face and neck (same case as depicted in Figure 34.4). Apart from the hanging mark, conspicuous excoriations and haematomas were located especially in the submental region (b). Nevertheless, the medical examiner who inspected the body erroneously assumed self-hanging.

- Significant signs of blood congestion in the head region in victims suspended freely and having the knot in the occipital region, haemorrhages of the anterior and lateral parts of the tongue due to compression between the dental arches [5,23,24,34,36], fracture of the cricoid cartilage [14], acute emphysema and haemorrhagic-dysoric syndrome of the lungs [8].
- Additional injuries in body regions apart from the neck (e.g. bruising or abrasion of the lips and around the nostrils due to smothering [35,48], blunt head trauma [21,25,41], haematomas from overpowering, bruising on the upper arms from gripping, lesions from tying, stabs and cuts [41]).
- Drag marks (e.g. grazes from pulling the body along the ground [36], contusions in victims with cardiac activity [13].
- No evidence of vitality, i.e. hanging during lifetime (absence of blood extravasations at the origin of the straight neck muscles and of so-called Simon's haemorrhages along the lumbar spine despite free suspension of the body).

In the relevant literature, some additional signs allegedly indicating homicidal hanging are mentioned such as tying [1,44] and gagging [41,53] as well as the interposition of textiles, fingers and strands of hair between the noose and the neck [28,41]. However, it has to be emphasized that there are also clear hanging suicides with self-applied fetters [2,4,16,18,30] or a gag in the mouth [2,26,37,49]. The same is true for hangings with garments, fingers or strands of hair under the ligature [7,16,35]. Likewise, the position of the victim (completely suspended, partly supported, semi-reclining, sitting, lying, standing, kneeling) is of dubious diagnostic value, as both suicidal and homicidal hangings can take place in any position [16]. A supracervical course of the noose is possible in both homicide and suicide cases [16,33]. Forensic traces potentially pointing to a masked crime (e.g. detection of a perpetrator's DNA on the hanging device [28,36], fibre evidence from crime scene investigation [36], a flow pattern of blood not consistent with the position of the hanging body [20,25,41]) cannot be discussed within the scope of this chapter.

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# 35 Suffocation during/after Anaesthesia or due to Medical Malpractice

*Burkhard Madea, Elke Doberentz and Frank Musshoff*

## 35.1 Adverse events and medical malpractice

*Burkhard Madea and Elke Doberentz*

### Definitions

Different institutions, including the European Council and the World Health Organization, have proposed their own specific definitions for adverse events and medical malpractice. Some general definitions are given here.

An adverse event (AE) is a noxious and unintended response.

- A preventable adverse event (PAE) is a noxious and unintended response that might have been prevented.
- A negligent adverse event (NAE) is a noxious and unintended response due to a break of duty of care.
- A NAE is an equivalent of medical malpractice.

In penal law, medical malpractice is defined as an AE (injury or harm) which is due to medical negligence. Medical negligence is defined as a preventable mistake due to a lapse of duty of care. Furthermore, there must be a causal connection between the mistake and the injury and, in most jurisdictions, this causal connection has to be proven without reasonable doubt.

In civil law, medical malpractice is defined slightly differently. The defendant (doctor) owes a duty of care to the plaintiff (patient). The doctor breaches the duty of care by failing to adhere to the standard of care expected, which is the quality that would be expected of a reasonable practitioner in similar circumstances. This breach of duty caused an injury to the patient.

Furthermore, for epidemiological research, a definition of medical error is of importance. Error may be an error of planning or execution. An error of execution is the failure of a planned action to be completed as intended; an error of planning is the use of a wrong plan to achieve an aim.

### Epidemiology

There are no clear data on the epidemiology of medical malpractice. In the United States, UK and Australia several studies have been conducted concerning AE, PAE and NAE but nearly exclusively on hospitalized patients. For ambulant medical care, data are scarce.

It is well known that only a small proportion of cases of harm or injury are on record and that the majority of cases do not come to light. This fact is often illustrated by an iceberg model of accidents and errors (Figure 35.1).

The German Alliance for Patient Safety carried out a systematic review of papers on the incidence of AEs, errors, etc. Studies fulfilling the following criteria were included:

- Original paper with data from January 1995 to December 2005.
- Data were collected on a well-defined reference group of patients.
- At least one of the following relevant criteria was checked:
  - Adverse event (AE)
  - Preventable adverse event (PAE)
  - Negligent adverse event (NAE)
  - Error
  - Near miss.

The paper had to contain a clear description of how the data were evaluated, and clear data on incidence such as proportion, ratio and incidence rates had to be evident. From more than 25 000 studies in PubMed and Embase, 151 studies from 25 countries with 7,686,166 patients fulfilled these criteria.

The review revealed a dependency of the frequency of reported AEs, PAEs and NAEs on the sample size: The larger the sample, the lower the frequency. Furthermore, there was no influence of the geographic origin of the study, so the results may be taken as representative for countries with a ‘western’ standard in health care.

Among hospitalized patients, AEs can be expected in 5–10 per cent, PAEs in 2–4 per cent, NAEs in about 1 percent and lethal outcome in about 0.1 per cent of cases. Based on epidemiological studies for hospitalized patients

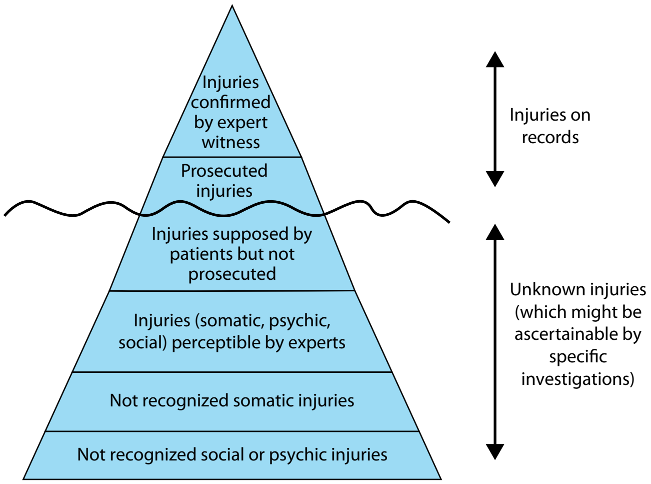
Injuries confirmed by expert witness
Prosecuted injuries
Injuries supposed by patients but not prosecuted
Injuries (somatic, psychic, social) perceptible by experts
Not recognized somatic injuries
Not recognized social or psychic injuries
Injuries on records
Unknown injuries (which might be ascertainable by specific investigations)

**Figure 35.1** Iceberg model of accidents and errors: Types of injury and probability of detection. Only a small proportion of injuries is on the records. (Source: Gutachten 2003 des Sachverständigenrates für die konzertierte Aktion im Gesundheitswesen Deutscher Bundestag, 15. Wahlperiode Drucksache 15/530.)

in Germany, this would mean that 880 000–1 750 000 AEs, 350 000–700 000 PAEs, 175 000 NAEs and 17,500 lethal cases per year would be expected. In contrast, in the official mortality statistics, less than 600 cases of death during medical interventions per year are registered. Thus, only a small proportion of these cases raise legal discussions. For most countries data on the frequency on medical malpractice claims are not available. For Germany it is estimated that only 1500–2000 cases a year are being investigated by the public prosecution department. These are mostly cases where death is thought to be due to medical malpractice, and the cause and manner of death have to be cleared by a legal autopsy. In penal law, it is estimated that one investigation by the prosecutor is performed on 60 000 inhabitants, with one report of the prosecutor on 90 000 inhabitants. On average, only eight cases per year are brought to a penal court, with four convictions and four stays of proceedings. Data on the frequency of medical malpractice claims in civil law are not available, but estimates are in the region of about 15 000 claims per year.

Every doctor is obliged to have liability insurance, although detailed data from the liability insurance companies are not available for research. One insurance company with 108 000 insured doctors reported about 4500 reportings a year, with a settlement of cases in 30 per cent, another 10 per cent going to a civil court and medical malpractice confirmed at court in 4 per cent of cases.

In Germany many claims of medical malpractice are dealt with at the arbitration committees of the medical councils, which were set up more than 30 years ago to make medical malpractice claims possible without applying to court. More than 10 000 cases per year are dealt with by the arbitration committees and patient claims are confirmed in 30 per cent of these. The data of the arbitration committees are presented once a year as a nationwide report, the Medical Error Reporting System (MERS).

There are about 12 000 claims at the arbitration committees a year nationwide, about 24 of which are justified and 76 which are not. More claims are against doctors working in hospital (72%) than those working in private practice. Leading problems as cause for complaints include gonarthrosis, coxarthrosis, fractures of the forearm, and fractures of the lower legs/ankle joint. The most frequent complaints against hospital doctors concerned surgical therapy, followed by postoperative care, diagnostic imaging and informed consent.

Although anaesthesia is a high-risk medical discipline, preventable injuries have been reduced by the introduction of standards for patient monitoring during anaesthesia. A review of insurance data indicates that approximately 1.5 claims are paid per 10 000 anaesthetic procedures, a conservative estimate of the incidence of preventable serious injuries associated with anaesthesia. In older retrospective studies, a death rate of 6.4 per 10 000 anesthetic procedures associated with anaesthesia was reported.

Standards of practice for patient monitoring during anaesthesia at Harvard Medical School can be found in Table 35.1. Important additional approaches for the reduction of anaesthesia morbidity and mortality are summarized in Table 35.2.

Important errors involving mechanical devices leading to asphyxiation are summarized in Table 35.3. Table 35.4 gives a classification of patient injuries due to anaesthesia. Many of the patient injuries are related to airway, ventilation or postoperative drug overhang. Table 35.5 analyzes respiratory and non-respiratory causes of 36 anaesthetic cardiac arrests between 1969 and 1988. Most respiratory of these anaesthetic cardiac arrests were preventable.

The complication rate for catheterization of the cervical veins depends highly on the skills and the experience of the doctor and is approximately 2 per cent. In addition to a pneumothorax, puncture of the arteria carotis interna is the most frequent complication (80–90%). In one fatal case a 68-year-old female patient with acute myeloblastic leukaemia died of suffocation after a failed catheterization of the internal jugular vein. After two attempts at placing the catheter, dyspnoea developed rapidly and intubation of the barely sedated patient failed. After a tracheotomy and failed ventilation attempts, the hypoxic women died – in the presence of nine doctors. The forensic autopsy revealed, among other things, two injuries to the arteria carotis with an extensive haematoma and an extratracheal position of the tracheal tube. Knowledge of anatomy, skilled performance and experienced handling of possible complications can minimize serious consequences of this routine intervention (Figures 35.2–32.5).

**Table 35.1 Department of Anesthesia, Harvard Medical School, Standards of Practice – I, Minimal Monitoring, 1985***

These standards apply for any administration of anesthesia involving Department of Anesthesia personnel and are specifically referable to preplanned anesthetics administered in designated anesthetizing locations (specific exclusion: administration of epidural analgesia for labor or pain management). In emergency circumstances in any location, immediate life support measures of whatever appropriate nature come first with attention turning to the measures described in these standards as soon as possible and practical. These are minimal standards that may be exceeded at any time based on the judgment of the involved anesthesia personnel. These standards encourage high-quality patient care but observing them cannot guarantee any specific patient outcome. These standards are subject to revision from time to time, as warranted by the evolution of technology and practice.

**Anesthesiologist’s or Nurse Anesthetist’s Presence in Operating Room**

For all anesthetics initiated by or involving a member of the department of anesthesia, an attending or resident anesthesiologist or nurse anesthetist shall be present in the room throughout the conduct of all general anesthetics, regional anesthetics, and monitored intravenous anesthetics. An exception is made when there is a direct known hazard, e.g. Radiation, to the anesthesiologist or nurse anesthetist, in which cause some provision for monitoring the patient must be made.

**Blood Pressure and Heart Disease**

Every patient receiving general anesthesia, regional anesthesia, or managed intravenous anesthesia shall have arterial blood pressure and heart rate measured at least every five minutes, where not clinically impractical.

**Electrocardiogram**

Every patient shall have the electrocardiogram continuously displayed from the induction or institution of anesthesia until preparing to leave the anesthetizing location, where not clinically impractical.

**Continuous Monitoring**

During every administration of general anesthesia, the anesthetist shall employ methods of continuously monitoring the patient’s ventilation and circulation. The methods shall include, for ventilation and circulation each, at least one of the following or the equivalent:

*For Ventilation:* Palpation or observation of the reservoir breathing bag, auscultation of breath sounds, monitoring of respiratory gases such as end-tidal carbon dioxide, or monitoring of expiratory gas flow. Monitoring end-tidal carbon dioxide is an emerging standard and is strongly preferred.

*For Circulation:* Palpation of a pulse, auscultation of heart sounds, monitoring of a tracing of intra-arterial pressure, pulse plethysmography / oximetry, or ultrasound peripheral pulse monitoring.

It is recognized that brief interruptions of the continuous monitoring may be unavoidable.

**Breathing System Disconnection Monitoring**

When ventilation is controlled by an automatic mechanical ventilator, there shall be in continuous use a device that is capable of detecting disconnection of any component of the breathing system. The device must give an audible signal when its alarm threshold is exceeded. (It is recognized that there are certain rare or unusual circumstances in which such a device may fail to detect a disconnection.)

**Oxygen Analyzer**

During every administration of general anesthesia using an anesthesia machine, the concentration of oxygen in the patient breathing system will be measured by a functioning oxygen analyzer with a low concentration limit alarm in use. This device must conform to the American National Standards Institute No. Z.79.10 standard.

**Ability to Measure Temperature**

During every administration of general anesthesia, there shall be readily available a means to measure the patient’s temperature.

*Rationale:* A means of temperature measurement must be available as a potential aid in the diagnosis and treatment of suspected or actual intraoperative hypothermia and malignant hyperthermia. The measurement/monitoring of temperature during *every* general anesthetic is not specifically mandated because of the potential risk of such monitoring and because of the likelihood of other physical signs giving earlier indication of the development of malignant hyperthermia.

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**Table 35.2 Important additional approaches to the reduction of anaesthesia morbidity and mortality**

1. Development of an effective programme of education, training and supervision.
2. Establishment of rigid and thorough risk management/quality assurance programmes.
3. Mandating use of protocols for anaesthesia machine checkouts and relief of one anaesthetist by another.
4. Installation of safe, up-to-date equipment and proper maintenance of it.
5. Provision of satisfactory anaesthesia workspaces and environments.
6. Maintenance of neat machine tops and worktables.
7. Implementing colour-coding of syringes.
8. Repeated examination of human error and recognition that none of us is exempt from it.
9. Recognition of the effect of fatigue and similar factors on vigilance.

*Source:* Reprinted by permission from Springer Nature License: Springer Nature. Monitoring instruments have significantly reduced anesthetic mishaps by Pierce EC Jr. *J Clin Monit* 1988;**4**:111–1145.

## 35.2 Death during patient-controlled analgesia: Piritramide overdose

### ■ *Burkhard Madea and Frank Musshoff*

Human errors play a crucial role in the safety of medical equipment. This is illustrated by a case of fatal respiratory depression associated with patient-controlled analgesia (PCA).

Piritramide (1-(3-cyano-3,3-diphenyl-propyl)-4-(1-piperidyl)piperidin-4-carboxamid) is a synthetic opioid indicated primarily for the management of postoperative pain (Figure 35.6). The drug is commonly applied via portable pumps in a PCA regime, providing safe self-administration of analgesics [60]. The steady-state plasma

**Table 35.3 Typical errors involving mechanical devices**

| |
|---|
| Endotracheal tubes dislodged. |
| Endotracheal tubes occluded (e.g. due to pulmonary secretions). |
| Endotracheal tubes pinched off. |
| Perforation of the trachea by an endotracheal tube. |

**Table 35.4 Classification of patient injuries[a]**

| | |
|---|---|
| **Airway/circuit integrity** | Disconnect and misconnect<br>Intubation error: oesophageal, bronchial obstruction |
| **Ventilation** | Operator error<br>Ventilator malfunction<br>Laryngospasm, bucking, straining |
| **Gaseous/intravenous agents** | Hypoxic mixture<br>Overdose<br>Excessive rate of administration<br>Wrong drug |
| **Miscellaneous intraoperative events** | Hypovolaemia<br>Aspiration<br>Pneumothorax<br>Dysrhythmias<br>Acid/base imbalance<br>Malignant hyperthermia |
| **Residual drug effects – post-anaesthetic** | Narcotics<br>Muscle relaxants<br>Halocarbons<br>Barbiturates and other |

*Source:* Reprinted by permission from Springer Nature License: Springer Nature. Monitoring instruments have significantly reduced anesthetic mishaps by Whitcher C, Ream AK, Parson D et al. *J Clin Monit* 1988;**4**:5–15.

[a] Cardiac arrest followed by restoration of cardiac function usually leads to mortality or severe morbidity.

**Table 35.5 Causes and preventability of 36 anaesthetic cardiac arrests, 1969–1988**

| | | **Preventable** | |
|---|---|---|---|
| **Cause** | | **Yes** | **No** |
| **Respiratory** | Unrecognized oesophageal intubation | 5 | |
| | Unable to ventilate after induction | 3 | |
| | Undiscovered ventilator disconnected | 2 | |
| | Dislodged endotracheal tube | 1 | |
| | Asthma with severe bronchospasm | | 1 |
| **Non-respiratory** | Inappropriate use/overdose of anaesthetic | 12 | |
| | Haemodynamic instability, arrest with induction | | 10 |
| | Hyperkalaemia after succinylcholine | 1 | |
| | Hypotension with spinal, delayed treatment | 1 | |

*Source:* Reprinted by permission from Springer Nature License: Springer Nature. Decreasing frequency of anesthetic cardiac arrests by Keenan RL. *J Clin Anesth* 1991;**3**:354–357.

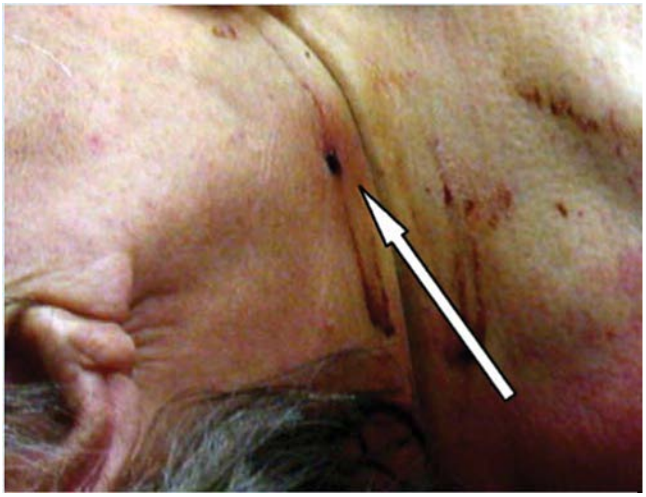

**Figure 35.2** Puncture for cerebral venous catheter site behind the right sternocleidoid muscle.

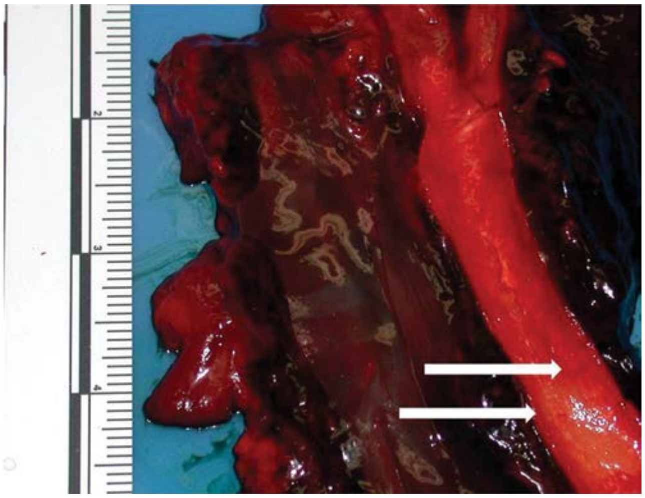

**Figure 35.3** Two small incised wounds of the right carotid artery.

concentration of piritramide necessary for 50 per cent of maximum analgesia (ED50) is between 2.9 and 29.8 ng/ml (mean 12.1 ng/ml) [56]. In comparison to other opioids, it has a quite slow onset of action (10–60 minutes) [51,73] and a long terminal elimination half-life of about 8 hours with a considerable variability with respect to the patient's age [38]. Therefore, piritramide has to be dosed carefully during long-term treatment to avoid accumulation that may lead to adverse effects [57].

Nausea and vomiting are described as rare side effects and haemodynamic stability is well maintained at therapeutic doses, although bradycardia and hypotension have been reported in some patients [58]. Furthermore, it has been demonstrated that piritramide displays unusually low respiratory depressant effects caused by a slow equilibration between the plasma and the site of action [37,79]. In general, respiratory depression associated with

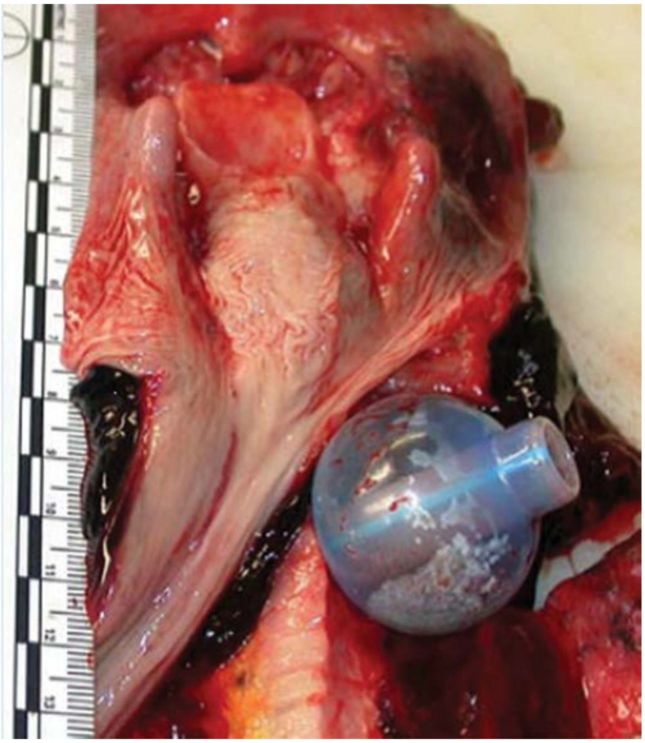

**Figure 35.4** Overview of the neck organs and lungs from dorsal with perforation of the pars membranacea of the trachea and extensive tissue haemorrhage.

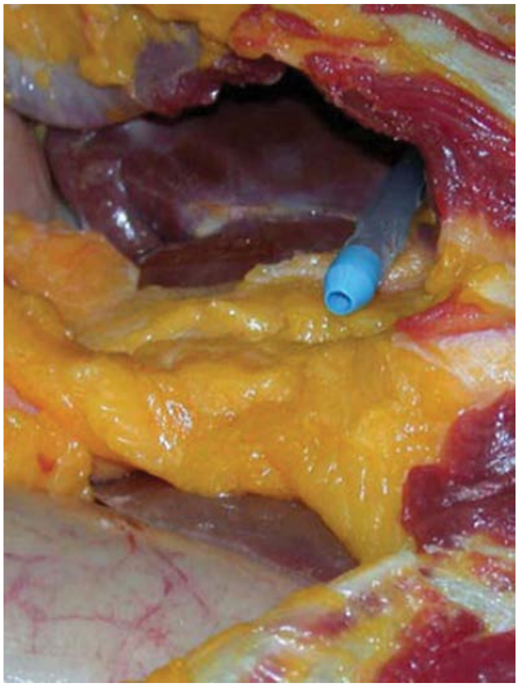

**Figure 35.5** Right Bülau-drainage in the abdominal cavity.

PCA has been described in a range of 0.33–0.5 per cent, less than reported from i.m. opioids with 0.9 per cent [46,64]. The most common side effects of piritramide appear to be a dose-related incidence of sedation [58].

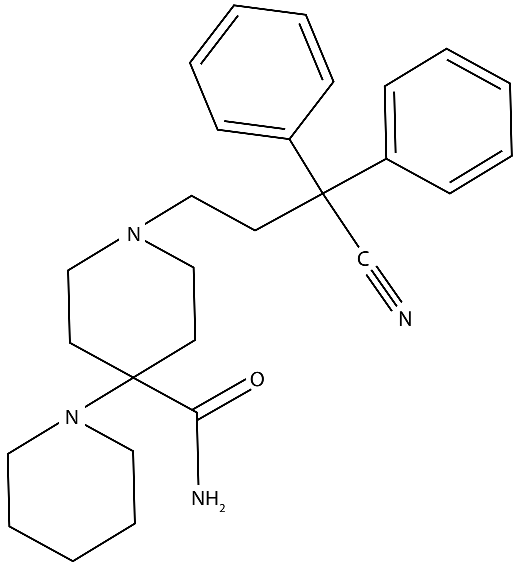

**Figure 35.6** Piritramide.

**Table 35.6** **Taxonomy of PCA safety hazards (according to Doyle [42])**

| Hazard | Reference |
|---|---|
| Use of wrong drug or wrong cartridge (e.g. 5 mg/ml morphine cartridge when a 1 mg/ml cartridge is required) | [77] |
| Accidental misprogramming (sometimes as a consequence of a hostile user interface) | [36,45,61,62,67,81] |
| False triggering (e.g. due to a short circuit in the PCA button) | [40,41,44,48,54,74] |
| False triggering by proxy (e.g. relatives pushing the PCA button because Granny is too sleepy to do it herself) | [43] |
| Drug accumulation in i.v. dead space under low flow conditions | [52,59,75] |
| Runaway fluid column due to 'siphoning' | [53,82] |
| PCA machine malfunction due to hardware failure (e.g. disengaged glass syringe) | [40,65,80] |
| Retrograde flow of PCA analgesic drug into a secondary i.v. set (e.g. antibiotics) due to a temporarily blocked i.v. catheter | [71,72] |
| Cracked syringe barrel | [76] |
| Anaphylaxis | |
| Extraordinary sensitivity to opiates (in elderly), renal failure, pre-existing sleep apnoea syndrome, background infusion or concomitant administration of sedative/ hypnotic medications resulting in unexpected respiratory depression | [46,49,64] |
| Reprogramming with criminal intent | |

In general, the efficiency and safety of PCA in the treatment of postoperative pain is well documented [47,78]. However, reports of safety hazards and deaths caused by fatal opioid overdose associated with PCA pumps are also described in the literature, and these are summarized in Table 35.6. Here, we report about a case where a patient died as a result of a drug overdose of piritramide.

## Case history

A previously healthy, 51-year-old male (height 164 cm, body weight 79 kg), underwent surgery of the left shoulder joint after tendon rupture of rotator muscles after a fall downstairs. Surgery was performed in general anaesthesia at 1:30 p.m. (premedication: clorazepate) induction: thiopentone, succinylcholine; maintenance: sufentanyl, isoflurane, vecuronium bromide as the patient had refused to receive a local anaesthesia (Winnie block). During anaesthesia, the patient had received cefazoline (antibiotic), metoclopramide (antiemitic), metamizole (analgesic) and ranitidine (stomach mucosa protection), as well as 3 × 500 ml of Ringer's lactate. The operation lasted for 85 minutes, the total anaesthesiologist's presence was documented for 200 minutes, and the patient was transferred to the recovery room after anaesthesia was ceased at 4:00 p.m. In the recovery room, vital parameters (arterial blood pressure, oxygen saturation) were documented to have returned to preoperative levels.

As the patient still complained about shoulder pain, PCA was initiated by the anaesthetist, and a piritramide (Dipidolor®) perfusor pump was started with a supposed concentration of 1.5 mg/h. According to the patient's record, the cartridge was changed at 11:50 p.m.; At that time, the patient was found still awake and free of pain, he reported to be satisfied with PCA and did not show any symptoms and signs of a potential piritramide overdosage.

The next day at 7:00 a.m., the patient was found dead in his bed by nursing staff. Resuscitation efforts initiated immediately remained unsuccessful; the patient was declared dead. The infusion pump was tested for hardware and software failures, but no errors were detected. The last bolus infusion was recorded between 3:00 and 3:59 a.m.

The autopsy and the histopathological examination revealed signs of operation of the acromioclavicular joint without signs of bleeding in this region or purulent abscesses, severe oedema of the brain (weight 1450 g) and lungs (left lung 620 g, right lung 850 g), blood without coagulation, urine in the bladder (740 ml) and congestion of all inner organs but no pre-existing diseases contributing to or as cause of death. Body fluids and tissues were collected at autopsy and stored at −18°C until analysis.

## Toxicological analysis

Various body fluids and organ tissues were assayed for ethanol and drugs of abuse (acidic, basic and neutral organic drugs) using routine methods including immunochemical procedures, the REMEDiHS (BIORAD, Munich, Germany) and liquid–liquid as well as solid-phase extraction procedures with further analysis by gas chromatography/mass spectrometry (GC/MS) and high-performance liquid chromatography with diode array detection (HPLC/DAD).

For the determination of piritramide, a HPLC-ESI-MS/MS procedure was used.

Following standard extraction procedures ranitidine, metoclopramine and desmethyldiazepam were identified in the urine sample by HPLC(REMEDi HS) and GC/MS. Quantitative analysis of a femoral blood sample by HPLC/DAD revealed insignificant results for these drugs in the following concentrations: desmethyldiazepam 0.08 mg/l; ranitidine 0.06 mg/l; metoclopramide 0.08 mg/l. A quantitative determination of piritramide was made by HPLC-ESI-MS/MS and the results are shown in Table 35.7. Considering the blood results, an overdose due to piritramide was proposed as cause of death.

Confronted with our results, investigations revealed that the PCA pump had been changed during a previous servicing from displaying mg/h to ml/h.The anaesthetist had entered '1.5' assuming mg/h, but had therefore actually applied 1.5 ml/h (which was therefore equivalent to 2.2 mg/h, given a concentration of the cartridge of 1.5 mg piritramide/ml). The change of displayed units (ml/h instead of mg/h) had been indicated by a red sticker on the back of the pump. In total, 61.5 ml (instead of 61.5 mg) had been infused, equivalent to 92.25 mg piritramide.

## Discussion

In the present case piritramide peripheral blood concentration of 0.1 mg/l exceeded the normal therapeutic range ($EC_{50} = 0.0121$ mg/l refers to severe pain using a visual analogue score rating of 75 on a scale from 0 (no pain) to 100 (worst imaginable pain). The highest concentrations were measured in the kidneys/urine and bile where, similar to other opioids, the drug had accumulated. Mean volume of distribution at steady-state concentrations ($V^{ss}$) is 4.7 l/kg, and this is the largest reported $V^{ss}$ of all opioids in clinical use, except for that of methadone (6.1 l/kg) [58]. The large $V^{ss}$ implies the difference in the drug concentration found in heart blood and peripheral blood and the phenomenon of drug redistribution, which is also described for methadone [39], but not for morphine ($V^{ss} = 3.3$ l/kg) [50,63] or for tramadol ($V^{ss} = 3.0$ l/kg) [69]. Considering the autopsy and histological findings, the results of toxicological analysis

**Table 35.7 Tissue distribution of piritramide in the present case**

| Specimen | Piritramide |
|---|---|
| Urine (mg/l) | 2.21 |
| Heart blood (mg/l) | 0.32 |
| Femoral blood (mg/l) | 0.10 |
| Kidneys (mg/kg) | 5.19 |
| Liver (mg/kg) | 1.89 |
| Bile (mg/l) | 2.51 |
| Brain (mg/kg) | 0.02 |
| Stomach content (mg/kg) | 0.48 |

were consistent with the assumption of a fatal overdose due to piritramide. Respiratory depression may be assumed to be the underlying pathophysiological mechanism.

Since the infusion pump had had 1.5 ml/h entered, erroneously intended as 1.5 mg/h, the patient had actually received 2.25 mg/h, causing a final overdosage of 1.5 times the intended dose of piritramide (total 92.25 mg piritramide instead of 61.5 mg), given a (correct) concentration of the cartridge of 1.5 mg/ml. This interpretation can also explain why the 4-hour limit of 50 ml (actually representing 75 mg) failed to protect the patient, in spite of keeping to the entered 4-hour limit of 50 ml as programmed (but not mg, as assumed by the physician). Regrettably, the change of units was not realized by the first physician, who initiated the pump in the afternoon, or by a second physician, who changed the cartridge shortly before midnight. The balance 61.4–92.25 mg is not so high; however, considering the inconspicuous autopsy and histological findings, the results of toxicological analysis were consistent with the assumption of a fatal overdose due to piritramide (exclusion of competitive causes of death with consideration of the circumstances of the death).

This case report is a powerful example illustrating that human errors play a crucial role in the safety of medical equipment. Based on a search of such incidents in various databases, mortality from user programming errors in PCA was estimated to be a low-likelihood event (ranging from 1:33 000 to 1:338 000), but relatively numerous in absolute terms (ranging from 65 to 667 deaths) [77]. By way of comparison, the authors calculated the likelihood of death from general anaesthesia to be 1: 200 000–1:300 000 [55]. As experts in anaesthesia, the authors recommended human factor engineering design principles to improve the safety of medical devices.

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## Death during patient-controlled analgesia: Piritramide overdose

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# 36 Entrapment and Incaprettamento

*Vittorio Fineschi, Matteo Scopetti and Emanuela Turillazzi*

'Asphyxia' is a term derived from Greek that translates literally as 'stopping of the pulse'. This term refers to a wide range of conditions and events in which there is inadequate delivery, uptake and/or utilization of oxygen by the body's tissues/cells, often accompanied by failure to eliminate carbon dioxide. Deaths due to asphyxia are quite common in the forensic setting, and they represent a significant percentage of all forensic autopsies [2].

Despite the need for a standardized classification [44], asphyxia of forensic interest can be substantially due to hanging, strangulation (manual, ligature), drowning, compression of the chest and abdomen, suffocation (environmental, smothering, choking, mechanical), and chemical substances (carbon monoxide, hydrogen cyanide, hydrogen sulfide). In addition, in some cases, the victim dies as a result of a combination of different mechanisms of asphyxia. Cases may, however, be difficult to diagnose when people (often family members) have interfered with the scene and removed material and devices or when no information about the death's scene is available. In fact, depending on the mechanism, individuals dying due to asphyxia may or may not have lesions, and to further complicate matters, the lesions of asphyxia may resemble those arising from other causes. Determination of the specific type(s) of asphyxia operative in a particular case, the cause and the manner of death are dependent on information elicited during the medicolegal death investigation – namely, history (circumstances), scene investigation and postmortem examination (including appropriate radiographic, toxicological and laboratory studies).

A number of terms can describe asphyxia that occurs when an individual is exposed to an atmosphere depleted in oxygen [44]:

- Asphyxia in a confined space.
- Entrapment or environmental suffocation (exclusion of oxygen).
- Suffocation.
- Death associated with exposure to gases in the atmosphere.
- Vitiated atmosphere.

Many authors in the forensic literature include in the category of confined space or vitiated atmosphere any kind of exclusion of oxygen as well as asphyxia from gases (carbon dioxide, carbon monoxide, methane and cyanide).

## Entrapment

According to DiMaio and DiMaio [17], entrapment and environmental suffocation are thought to be different asphyxial entities from suffocating gases and chemical asphyxia.

Following this restrictive interpretation, in this chapter we will focus on suffocation by entrapment or environmental hazard, both sharing the fact of inadequate oxygen in the environment. A confined space is any place which is completely or partly enclosed and where it is foreseeable that hazardous substances or conditions either inside it or nearby may cause a risk of the following occurring:

- Loss of consciousness from poisonous gases or lack of oxygen.
- Asphyxiation by free-flowing solids.
- Drowning in an increasing level of liquid.
- Serious injury by fire or explosion.

The hazards found in any confined spaces are represented by the process taking place in the confined space, by the eventual material being stored or used in the confined space, and by the effect of the external environment. Oxygen deficiency occurs from biochemical reactions which displace or consume oxygen from a confined space. Normally, ambient air has an oxygen content of 21 per cent. When the oxygen level drops, physiological responses include increased breathing volume and accelerated heartbeat. If the oxygen shortage increases (14–16%), poor muscular coordination, rapid fatigue and intermittent respiration occur. Between 6 per cent and 10 per cent oxygen victims experience nausea, vomiting, inability to perform and unconsciousness. At concentrations lower than 6–8 per cent there is loss of consciousness and death in minutes. Furthermore, toxic gases (i.e. carbon monoxide, hydrogen sulfide, etc.) may be present in particular confined spaces, mostly working places, and can play a significant role in causing death. Finally, it has to be remembered that physical hazards, including extreme hot or cold temperature, activation of electrical or mechanical equipment, falling objects and flammable atmosphere can cause further injury to victims in confined spaces.

These deaths are almost exclusively accidental in nature. In entrapment, individuals find themselves trapped in an airtight or relatively airtight enclosure and they are incapable of self-extrication. Generally, an entrapped person is blocked between the chest and shoulder level; however, any situation in which the subject is unable to get free and he/she is in a confined space can cause asphyxiation. Initially, there is sufficient oxygen to breathe. However, as respiration continues, the victim exhausts the oxygen and asphyxiates.

Although this kind of death is almost always accidental, suicide and homicide by entrapment can occur, the latter being the result of direct actions or negligence.

Accidental entrapment is frequent in childhood when exploratory behaviour is mainly directed towards the physical world. Very young children are naturally curious. They learn about the world by physically interacting with things and the environment around them. Furthermore, they are unable to judge risks appropriately. As a result of this, they are at high risk for accidents that often occur because the children are not capable of handling a hazardous situation either physically or cognitively [8]. The literature is rich in anecdotal reports of lethal entrapment in childhood, showing that younger children may be trapped by furniture or by industrial equipment while playing, while older children may be trapped under or by motor vehicles in similar circumstances to adult traumatic asphyxial death [9,10,27]. Entrapment by an automobile door has been described in adults and occurs when an occupant, usually the driver, has leant out of a vehicle which has then rolled forward and impacted the opened door. The victim is unable to self-extricate as forward movement of the vehicle wedges them firmly between the door, frame and/or another object [7,11].

In the adult population, worker fatalities related to confined spaces are quite common. The riskiest industries include manufacturing, agriculture, construction, transportation/communication/public utilities and mineral/oil/gas. Confined spaces such as storage tanks, silos, vats/pits, digesters, wells, ventilation ducts and sewer manholes are frequently involved in fatal incidents. Confined spaces are found in many industrial and agricultural settings. Agricultural confined-space entrapment fatalities involving grain, forage and manure storage structures and agricultural transport vehicles are not uncommon [25,26,29,42].

A complete forensic death investigation in fatalities related to entrapment is of crucial importance as, very often, external and internal cadaveric signs are non-specific.

As a fundamental part of the forensic investigation, a thorough, accurate inspection and description of the scene are critical to ensure completeness of the investigation process. Any items of forensic evidence should be documented by photographs and clearly labelled with the date; all information obtained during the scene investigation should be documented in the written report. In entrapment-related death, a thorough and complete investigation commonly leads to a high suspicion regarding the cause and manner of death prior to the autopsy itself (Figure 36.1a,b). In addition, by viewing the body in the context of its surroundings, the forensic pathologist is better able to interpret certain findings at autopsy (Figure 36.2a–c) [16].

## Corpse examination

Conventional radiography and multidetector computed tomography (MDCT) may be used as adjuncts to autopsy; however, in entrapment deaths, no specific imaging findings are described to aid in the diagnosis of the manner of death. Concurrent traumatic lesions and/or mechanical obstruction of the respiratory tree in the event of engulfment (events in which an individual is submerged by any kind of material) may be detected [32].

External examination of the victim's body often reveals non-specific asphyxial signs, represented by petechiae (tiny pinpoint haemorrhages). Petechiae may be present in tissues and mucous membranes, and they are mostly visible at bulbar and/or palpebral conjunctiva, face or neck. According to the literature, formation of petechiae is related to the combined effects of increased cephalic venous pressure and hypoxic damage to endothelial cells [18]. Although petechiae are very frequent in asphyxial deaths,

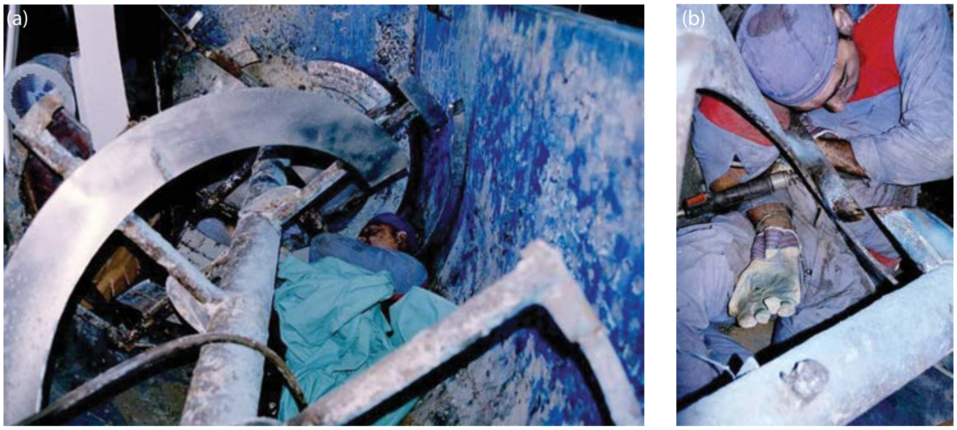

**Figure 36.1** (a) Scene of a death due to entrapment; (b) position of the body in relation to the surrounding environment.

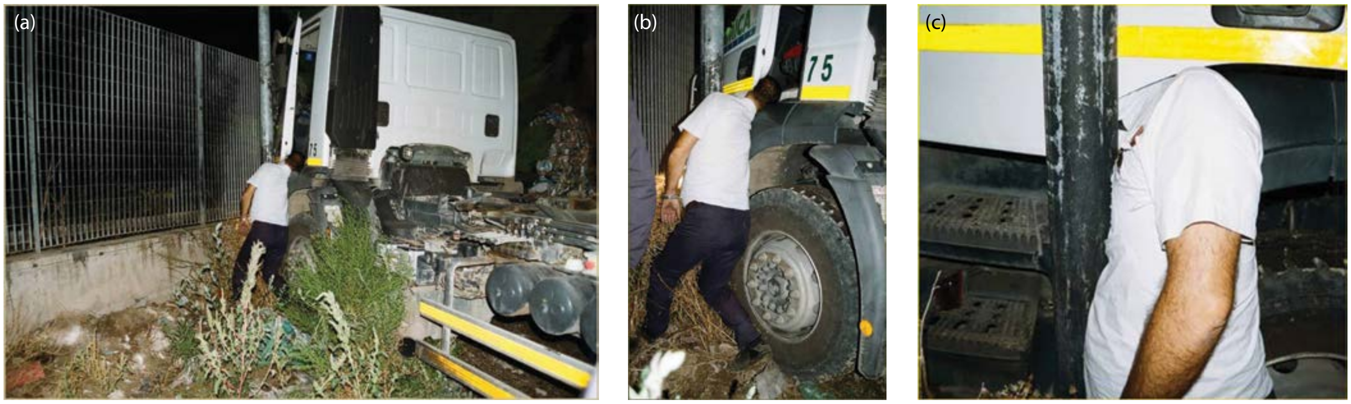

**Figure 36.2** (a) Scene of a death due to entrapment; (b, c) details of the position of the body compared to its surrounding.

they are not pathognomonic of this manner of death, thus representing non-probative evidence. Labial and subungual cyanosis may be present.

Pulmonary oedema with or without haemorrhage, atelectasis and interstitial emphysema are common macroscopic and microscopic lesions in asphyxial deaths but they also occur with a myriad of other conditions. Semi-quantitative evaluation of morphological parameters in lung specimens has been proposed as a useful supplementary histological criterion to support the diagnosis of asphyxia [15,23]. Visceral congestion, tissue oedema, petechial haemorrhages of various internal organs, and fluidity of blood are the most frequent, although uncharacteristic, internal morphological findings. In conclusion, specific external and/or internal signs do not exist in asphyxial deaths and the classic signs (cyanosis, fluidity of the blood, engorgement of the right ventricle, visceral congestion and petechiae) are often referred to as 'the obsolescent quintet' [6,34,36,40,41,43].

## Histopathology

The histopathologically detectable effects of oxygen deficiency in the inner organs and tissues depend on several factors, such as the effectiveness and the duration of the asphyxial process and the sensitivity of the tissues. Asphyxial deaths are caused by the failure of cells to receive and/or use oxygen. The brain is very sensitive to oxygen deprivation, and it is the organ mostly affected in all types of asphyxial death. Brain specimens may show the typical aspects of hypoxic damage. Neuronal hypoxic–ischaemic injury is generally characterized by cell bodies being variably shrunken. Cytoplasm tint varies from vivid pink to reddish with H&E stains. The nucleus is shrunken. An irregular contour is present in some cells, related to swollen astrocyte processes irregularly indenting the neuronal cell body and dendrites. These changes evolve in further shrinkage of the neuronal nucleus and cytoplasm and in dark red staining of the cytoplasm itself (Figures 36.3–36.5).

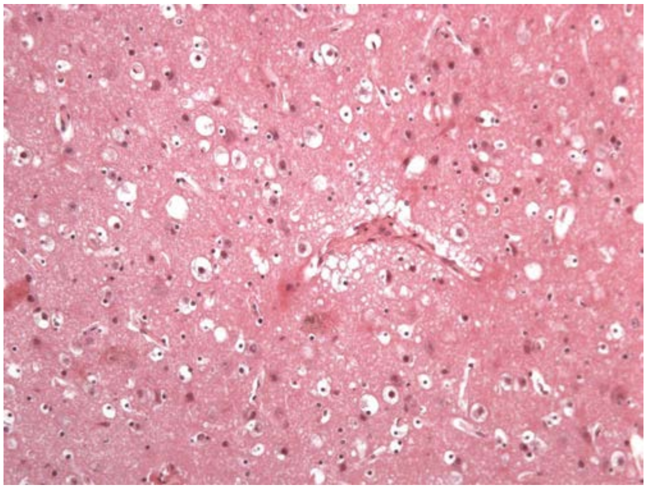

**Figure 36.3** Asphyxial death. A diffuse perineuronal oedema is evident as pericellular haloes containing amorphous material (H&E, ×100).

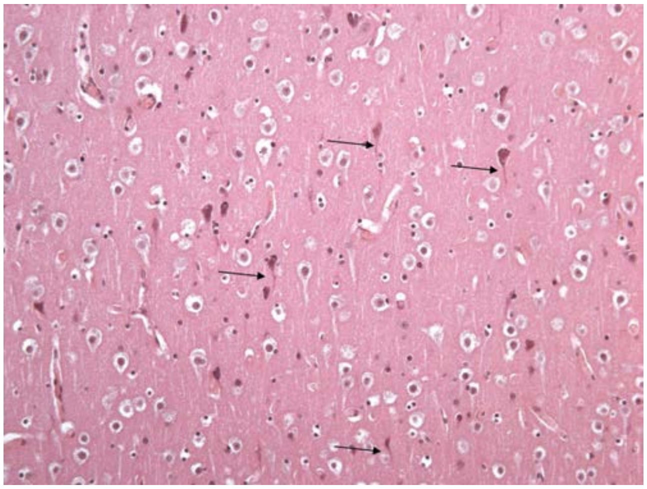

**Figure 36.4** Same case as Figure 36.3. Neuronal changes (cytoplasmic eosinophilia and cell shrinkage) are evident. At higher magnification note the corkscrew- like apical dendrites (arrows) in shrunken neurones with red basophilic cytoplasm (H&E, ×100).

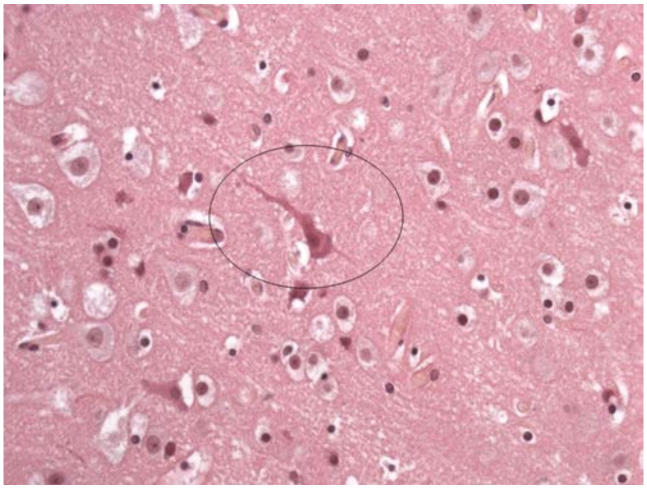

**Figure 36.5** Perineuronal oedema with a halo of amorphous material surrounding the cells. A dark red neurone with corkscrew-like dendrite is evident (H&E, ×200).

The nucleus is shrunken, often triangular, and pyknotic (Figures 36.6 and 36.7). Changes of the Golgi complex and endoplasmic reticulum, the persistence of free ribosomes, breakdown of granular endoplasmic reticulum, and an increase in nuclear density are described ultrastructurally.

The hypoxic–ischaemic neuronal changes continue to evolve to further shrinkage of the neuronal nucleus and cytoplasm and to dark pink–red staining of the cytoplasm. After at least 24 hours of survival, further hypoxic–ischaemic neuronal changes include decreased cytoplasmic volume, increased homogenation and loss of staining intensity of the cytoplasm. The nuclei become more eosinophilic and appear to blend in with the surrounding cytoplasm (Figures 36.8 and 36.9).

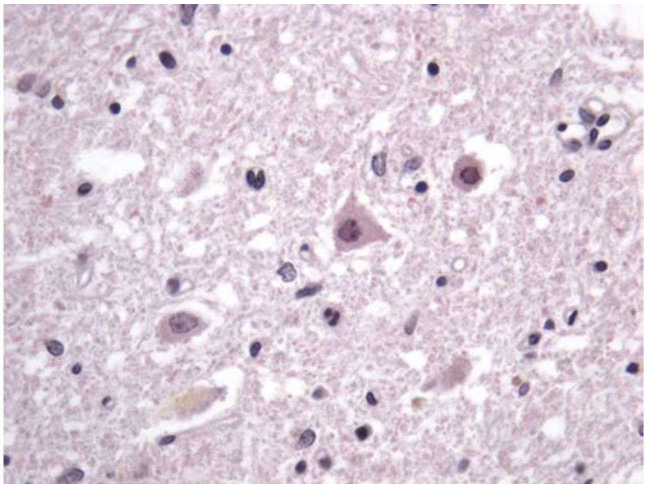

**Figure 36.6** Hypoxic nuclei are generally shrunken; nuclear pyknosis with indistinct nucleoli is another typical change in hypoxic brain insult (H&E, ×200).

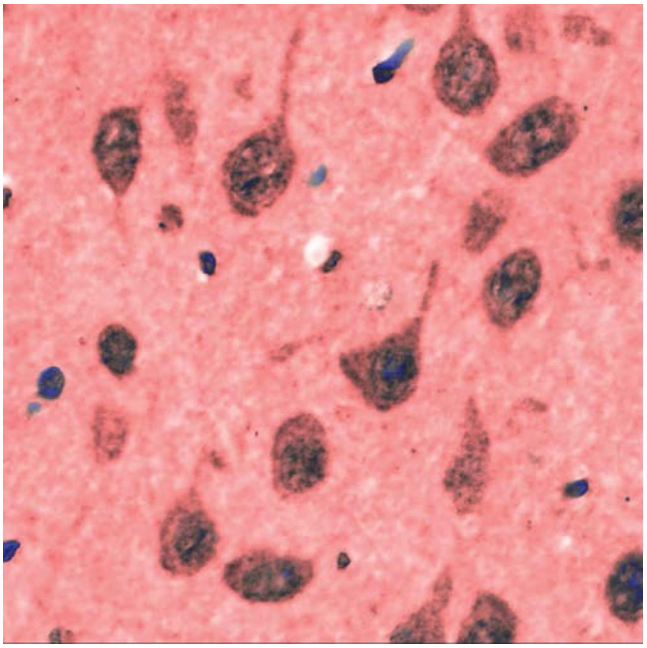

**Figure 36.7** Confocal laser scanning microscopy of nuclear changes (pyknosis with less distinct nucleoli) (×400).

These changes are particularly likely to be seen in neurones that are more sensitive to hypoxia (hippocampal neurones, pyramidal neurones in the cerebral neocortex, cerebellar Purkinje cells) (Figures 36.10 and 36.11).

Other organs findings, which do not themselves provide definitive proof, may include polivisceral stasis and intraparenchymal haemorrhages, mainly cerebral and splenic (Figures 36.12 and 36.13).

In the lungs, acute emphysema may be histologically detected in many cases of acute or subacute asphyxia (Figure 36.14).

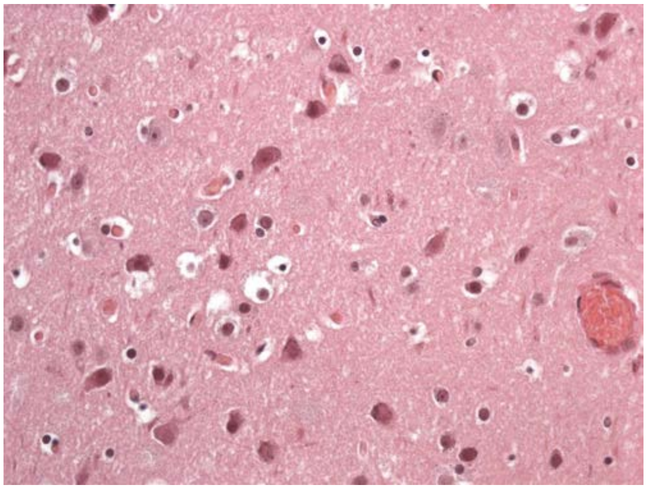

**Figure 36.8** Homogenizing cell changes, demonstrating cells with indistinct cell membranes and organelles. Pronounced cytoplasmic eosinophilia, loss of definition of cell membranes and pyknotic nuclei are typical hypoxic neuronal changes (H&E, ×200).

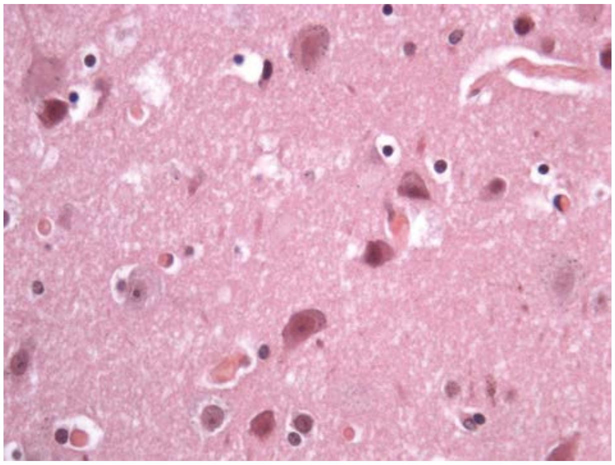

**Figure 36.9** At higher magnification the changes shown in Figure 36.8 are more clearly detectable (H&E, ×200).

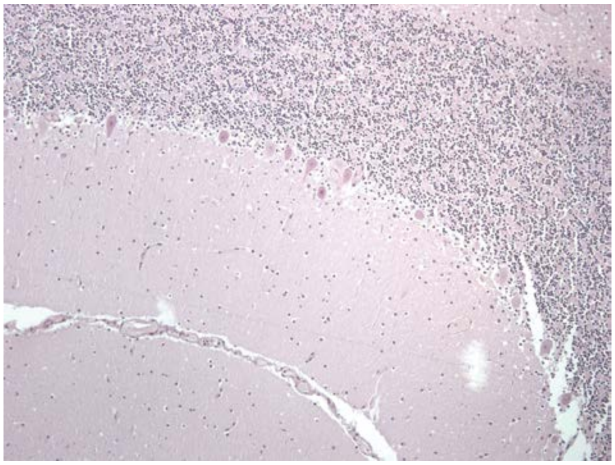

**Figure 36.10** Hypoxic change in cerebellar Purkinje cells. The cerebellum is particularly sensible to hypoxic insult. Eosinophilic Purkinje cells are evident (H&E, ×40).

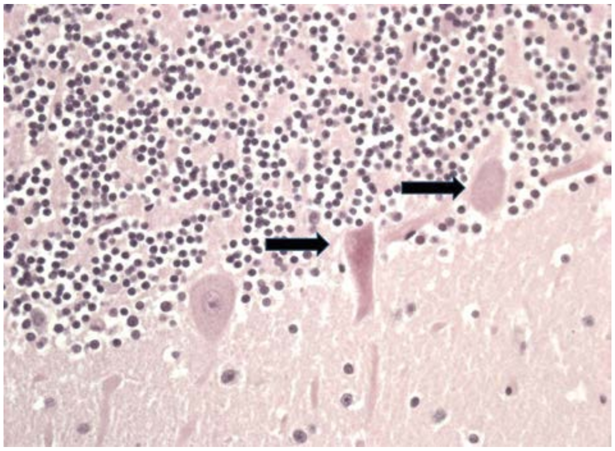

**Figure 36.11** High-power view highlights the eosinophilic Purkinjie cells with smudged and piknotic nuclei (arrows). In contrast, the normal Purkinje cells have plump large, vesicular nuclei and prominent nucleoli (H&E, ×200).

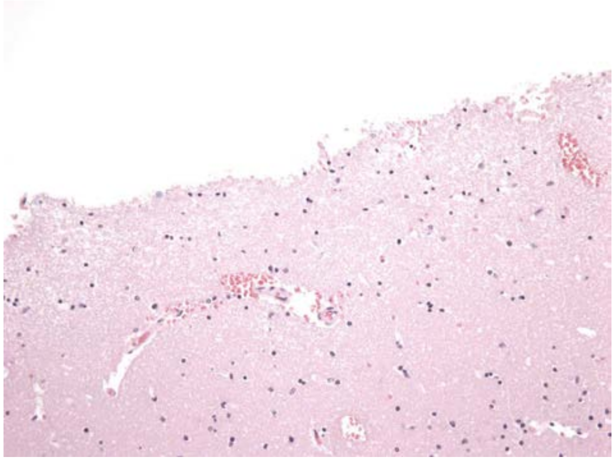

**Figure 36.12** Death due to hanging. Intracerebral small haemorrhages (H&E, ×40).

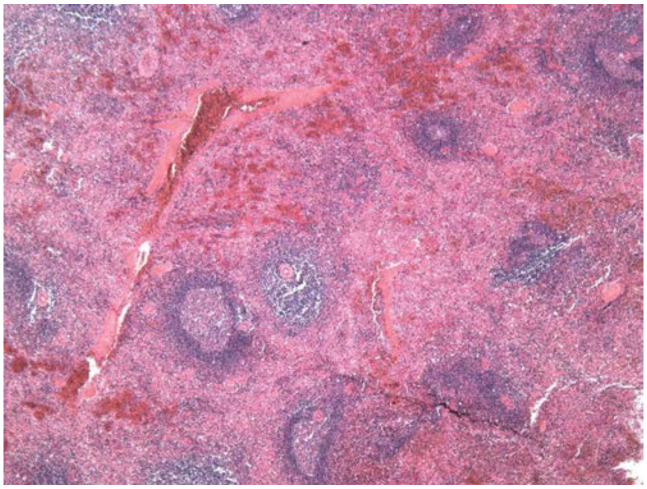

**Figure 36.13** Spleen intraparenchymal haemorrhages (H&E, ×40).

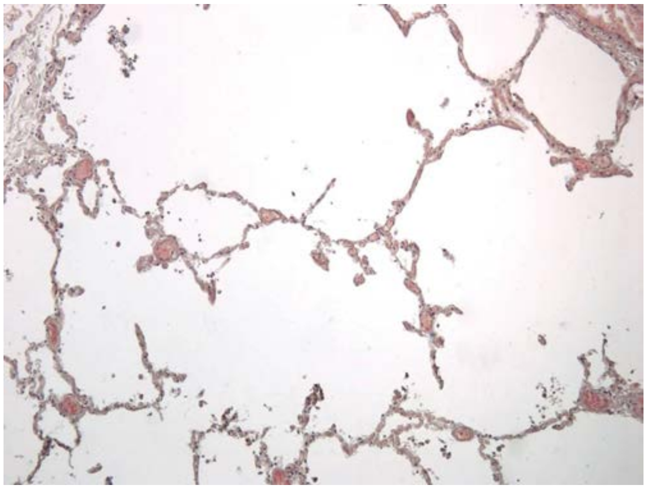

**Figure 36.14** Acute emphysema with bullous alveolar expansion. Pronounced vessel congestion is also detectable (H&E, ×40).

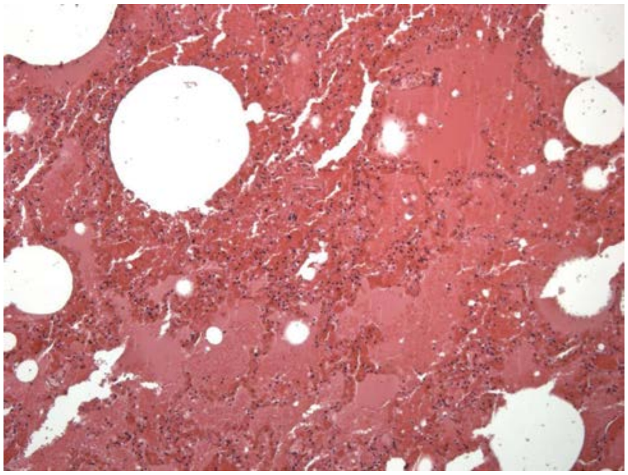

**Figure 36.15** A large amount of intra-alveolar haemorrhage in an asphyxial death; alveolar spaces are filled with crammed erythrocytes (H&E, ×100).

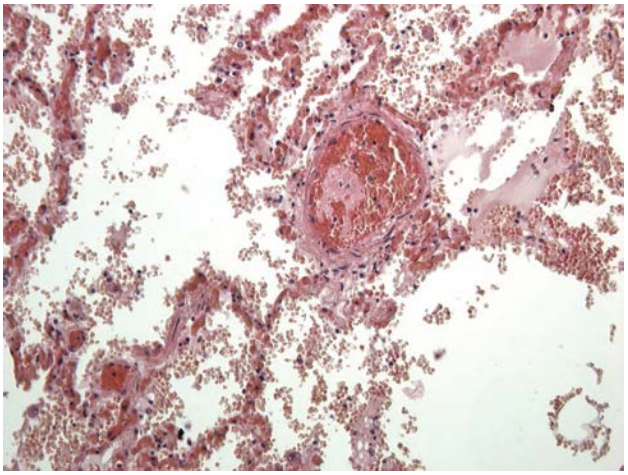

**Figure 36.16** Intra-alveolar haemorrhage in an asphyxial death (H&E, ×100).

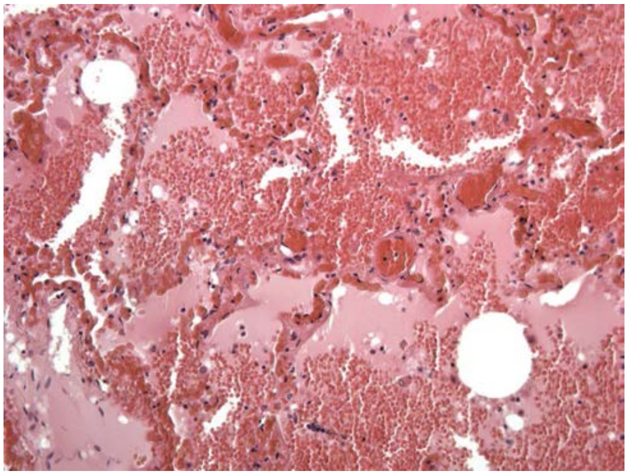

**Figure 36.17** Interstitial haemorrhages, alveolar haemorrhages and oedema (H&E, ×100).

Interstitial and alveolar haemorrhages are also found and they may be very extensive in the lungs (Figures 36.15–36.17).

Particular attention has been given to the demonstration of macrophages and giant cells in pulmonary tissue following asphyxia, mostly considered as a marker of protracted, slow asphyxia [50]. It has been proposed that quantification of macrophages and giant cells could differentiate between slow and acute asphyxia [49]; however, the issue is still debatable [24]. Evidence of oedema and of the presence of alveolar spaces filled with macrophages and giant cells is detected at histological observation in lung tissues of entrapped victims (Figures 36.18–36.23).

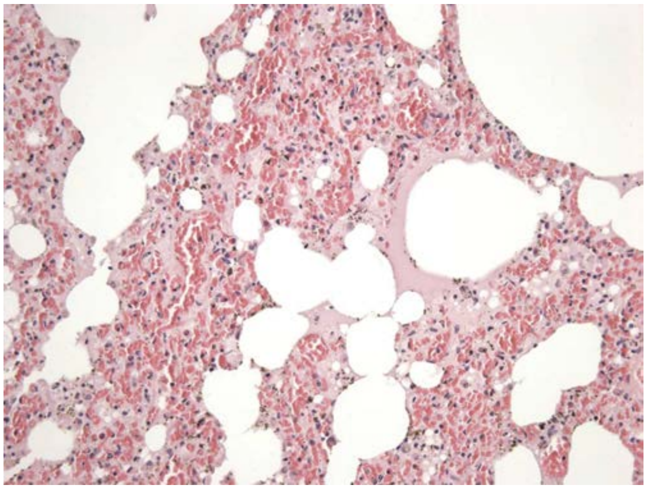

**Figure 36.18** Death due to hanging. Pulmonary alveoli with macrophages, giant cells and erythrocytes are evident (H&E, ×100). Following slow asphyxiation, abnormal migration of macrophages and giant cells into the alveolar spaces may be observed.

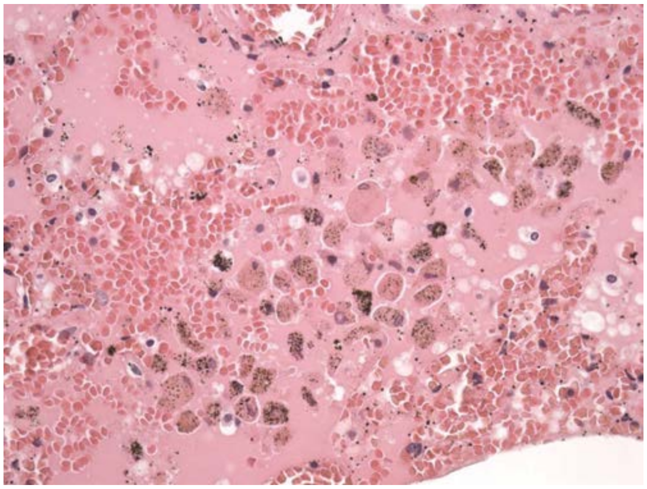

**Figure 36.19** At higher magnification intra-alveolar phagocytes and giant cells in the oedematic regions of the lungs can be seen (H&E, ×200).

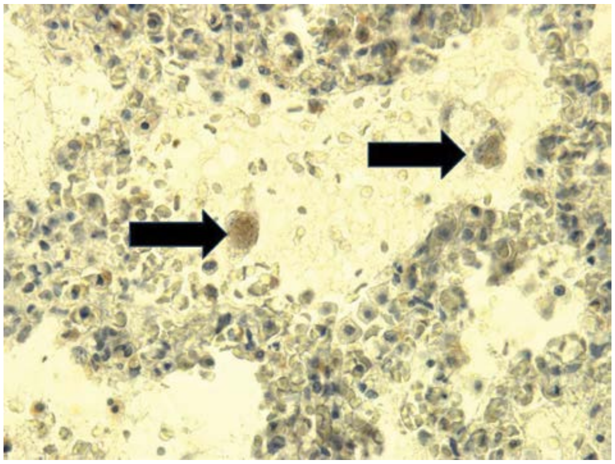

**Figure 36.20** Immunohistochemistry. Intra-alveolar phagocytes in the oedematic regions of the lungs (H&E, ×200).

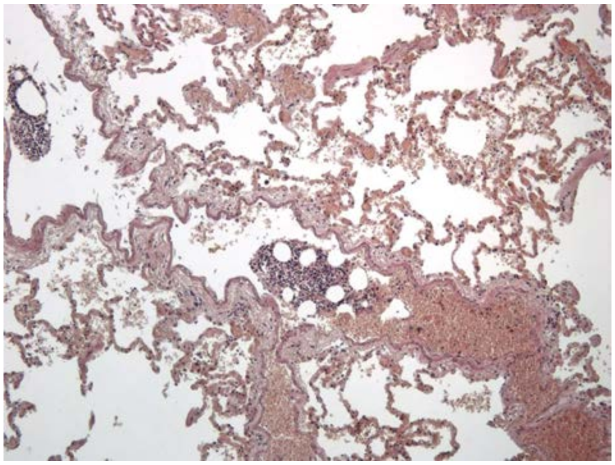

**Figure 36.21** Asphyxial death due to suffocation. Along with the intense vessels, congestion evidence of bone marrow embolism is present. An embolus with rich cellularity mixed with adipose microspherules is clearly evident (H&E, ×40).

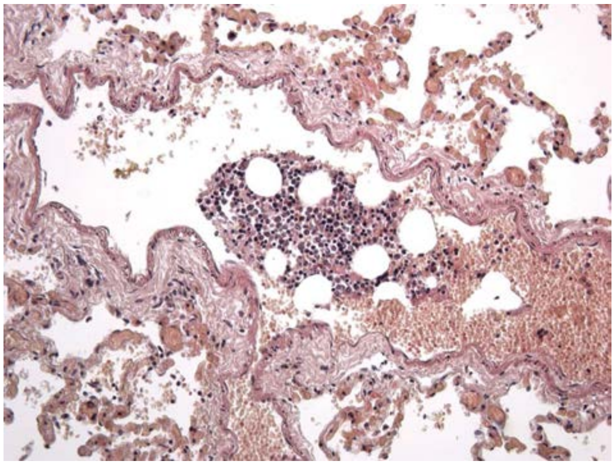

**Figure 36.22** At higher magnification better evidence of the clusters of haematopoietic cells admixed with adipocytes can be seen (H&E, ×100).

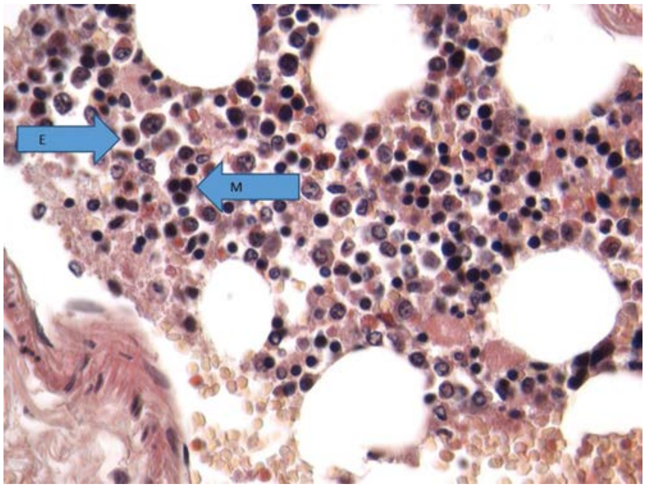

**Figure 36.23** Multiple cellular lineages present in the bone marrow embolus (H&E, ×400). E = erythroid; M = megakaryocytic, granulocytic and lymphocytic.

## Immunohistochemistry

Immunohistochemistry may be a very useful tool in the investigation of asphyxia deaths. As in other types of asphyxia death, immunohistochemically detectable changes in pulmonary tissue are considered of great importance, since lungs show several adaptive pathways to hypoxia. In particular, studies have focused on hypoxia-inducible factor 1 (HIF-1), a transcriptional activator that is expressed in response to cellular hypoxia and mediates multiple cellular and systemic homeostatic responses to hypoxia. Evidence from the literature has revealed an exponential increase in HIF-1α levels at $O_2$ concentrations less than 6 per cent (~40 mmHg); and experimental animal models have shown increased HIF-1α and HIF-1β mRNA levels in the lungs of mice exposed to acute or chronic hypoxia [37,38]. Yu et al. [52] demonstrated that HIF-1 expression was tightly coupled to $O_2$ concentration *in vivo* and the involvement of HIF-1 in the physiological and pathophysiological responses to acute hypoxia in the lungs. In most adult tissues, $O_2$ concentrations are in the range of 3–5 per cent and any decrease occurs along the steep portion of the dose-response curve, allowing a graded response to hypoxia [45]. HIF senses and coordinates cellular responses to hypoxia (Figures 36.24 and 36.25). Due to these observations, HIF-1α has been proposed as an useful tool in the assessment of asphyxial deaths and it is expressed in small-, medium-, and large caliber lung vessels in lung specimens of subjects dead due to asphyxia [12].

Mast cells (MCs) could be investigated in asphyxia deaths since their activation has been demonstrated in the development of hypoxia-induced inflammation [33,46]. Recent studies have confirmed that even short periods (few minutes) of lack of oxygen in humans (acute asphyxia/hypoxia) result in large quantities of MCs being rapidly recruited in the lungs; moreover, upon being recruited, they are not distributed evenly throughout the tissue, with

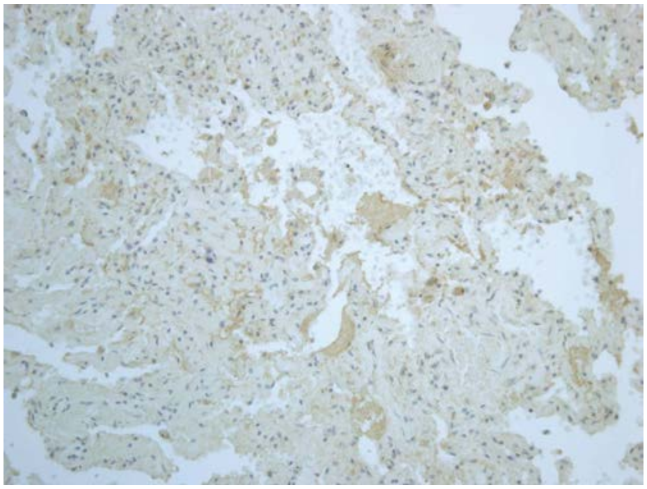

**Figure 36.24** Asphyxial death. Strong immunopositivity to anti-HIF-1α antibody, mainly with a perivasal disposition (×100).

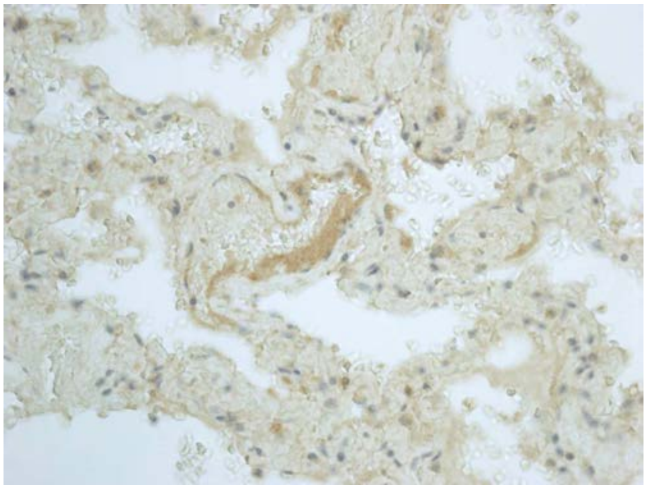

**Figure 36.25** Strong immunopositivity to anti-HIF-1α antibody viewed at higher magnification (×200).

a higher number of MCs being observed in perivascular areas than in control cases [12].

Finally, death due to entrapment in small, enclosed spaces is most likely due to oxygen deprivation, but hyperthermia and heat stroke also need to be considered [1,13,14]. Immunohistochemical investigation of markers of heat shock (heat shock proteins HSP90, HSP70 and HSP27) should be performed when circumstantial data suggest a potential causal inference of such mechanisms [20].

## Incaprettamento

'Incaprettamento' is a homicidal ritual practised by the Italian mafia. Incaprettamento is performed with a rope, one end of which is tied in a noose and placed around the victim's neck, while the other is used to secure the victim's ankles behind the back. Death is caused by self-strangulation when it becomes impossible to maintain the legs in this imposed position. However, often, victims are killed by strangulation, and the binding of the wrists and ankles in the typical position is performed postmortem [19]. The bindings of limbs or the rest of the body after death may be used by the assailant to facilitate transportation and disposal of the victim [51]. Unusual cases of 'incaprettamento' which showed great similarities to typical incaprettamento have been anecdotally described [21,39].

In such cases of death, medical pathologists may be in a quandary regarding the ante- or postmortem nature of the ligature marks. The traditional gross and microscopic examination of the lesions may be unreliable and may mislead the forensic pathologist in drawing conclusions as to whether they were due to lifetime or postmortem injuries.

Classic research in forensic medicine attached great value to haemorrhages as a sign of vital reaction in very different kinds of injury. Many mechanical actions, such as blunt force with contusion and rupture of the tissues including the blood vessels, or a sharp force, with severance of the blood vessels, may occur and explain the splitting of the vascular walls and blood escape into the surrounding tissue which can present a great variety of forms. Haemorrhage can occur without displacement of the tissue; in these cases the histological finding is the presence of corpuscular components of the blood detectable outside the vessels within the tissue concerned. Sometimes the blood extravasation is associated with displacement and laceration of the tissues themselves (Figure 36.26).

Our increased knowledge of the phases of skin-wound healing [30,31,35] has led to significant progress in the assessment of wound vitality in the forensic field.

Since an inflammatory reaction is a common response of the tissues to various forms of insult, the detection of

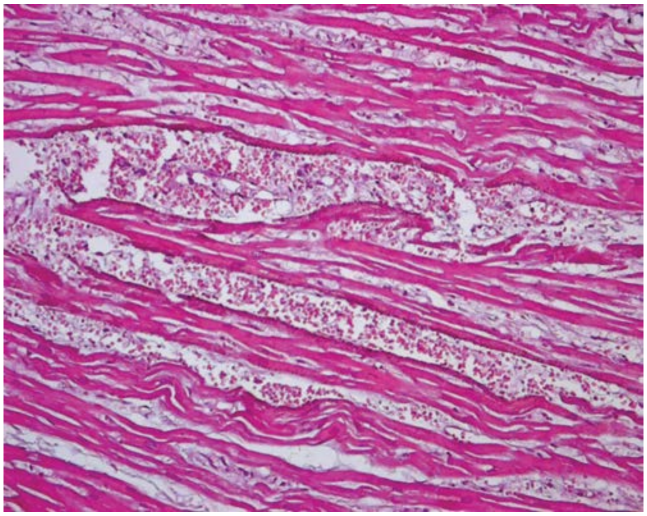

**Figure 36.26** Haemorrhages into the muscular tissue. The histological finding is the presence of corpuscular components of the blood detectable outside the vessels within the tissue concerned (H&E, ×100).

the inflammatory cascade which comprises both cellular and molecular components may be a useful diagnostic tool in detecting the vitality and the age of a lesion. When a traumatic injury occurs, an inflammatory response follows that involves enzyme activation, mediator release, inflammatory cell migration, tissue breakdown and repair. The major inflammatory cells that are activated and accumulate within the tissues are blood-derived leucocytes and macrophages. Positive reactions, defined as the presence of more than 10 cells outside the areas of bleeding, are first detectable in skin wounds after about 20–30 minutes [3,28]. In experimental studies on leucocyte recruitment during local inflammation, neutrophils were the predominant cell type at all time points (0, 2, 4, 6, 14, 24, 48 and 72 hours) after the injection of an inflammatory stimulus [22]. However, Saukko and Knight [43] underline that margination of polymorphonuclear leucocytes in dilated small vessels near a wound is not a reliable sign of antemortem infliction, as leucocytes may congregate for many hours after death, especially in skin and around aspirated material in the lung. The number of neutrophils decreases with increasing postmortem interval but these cells are also present (in reduced amounts) in some lesions aged more than 1 month [3]. Neutrophil infiltration can be identified with routine H&E stain (Figure 36.27).

As expected, CD15 antigen is an adequate parameter in detecting the local neutrophil response to trauma even in the absence of evident gross signs (such as ecchymosis, oedema, compressions, ligature marks, burns, etc.) (Figure 36.28). Confocal laser microscope allows a greater definition of the cellular morphology (Figure 36.29).

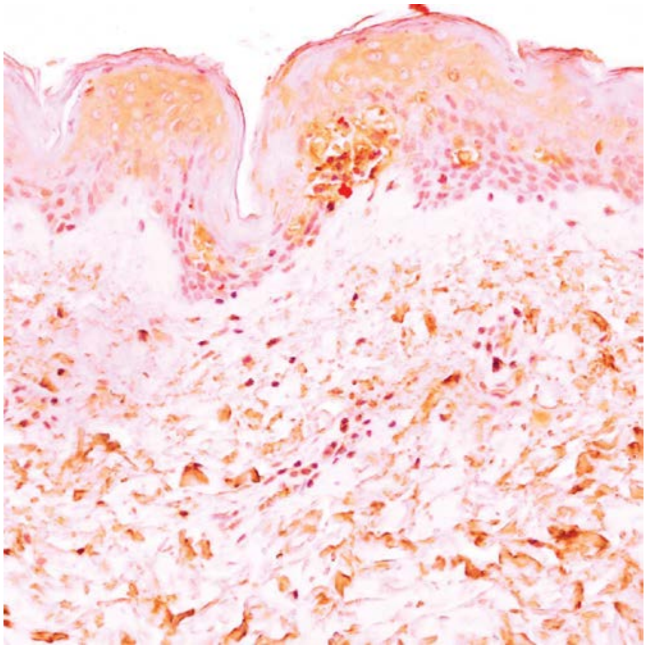

**Figure 36.27** Polymorphonuclear leucocytes in dilated small vessels near a wound (×200).

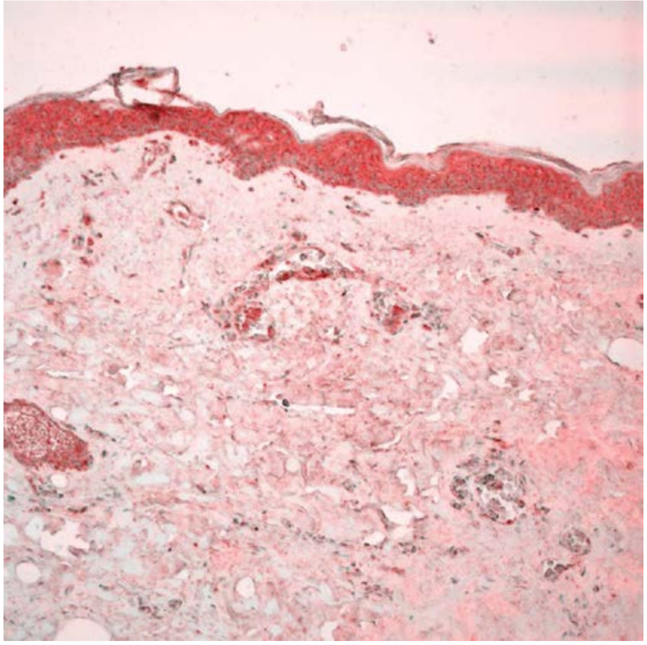

**Figure 36.28** CD15 (black reactions) detecting the local neutrophils response to trauma even if there are no evident gross signs (×40).

Macrophage infiltration is a regular finding after a postmortem interval of 15 hours or more. Fibrin appears in the wound within few minutes, but this also occurs in postmortem injuries [43].

MC activation in the site of trauma is a well-known vital reaction. Because MCs contain a variety of potent mediators, including histamine, heparin, proteinases, leukotrienes and

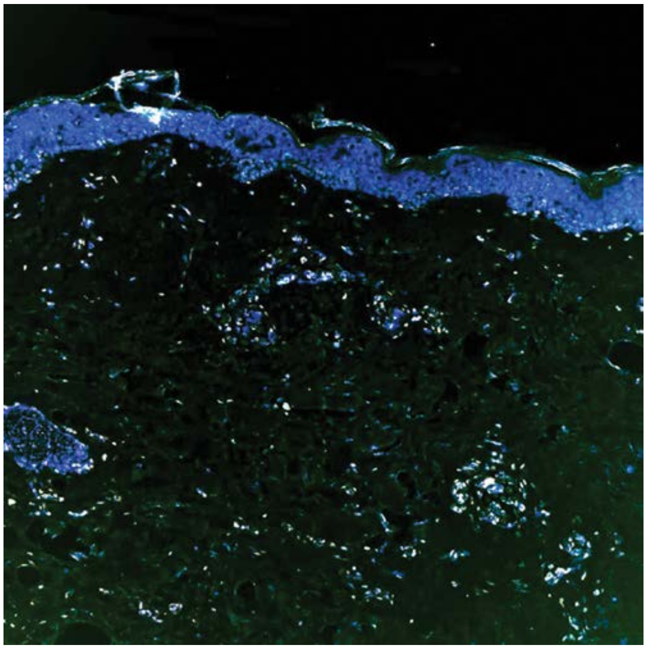

**Figure 36.29** Confocal laser microscope: CD15 (bleu reactions) detecting the local neutrophil response to trauma even if there are no evident gross signs (×40).

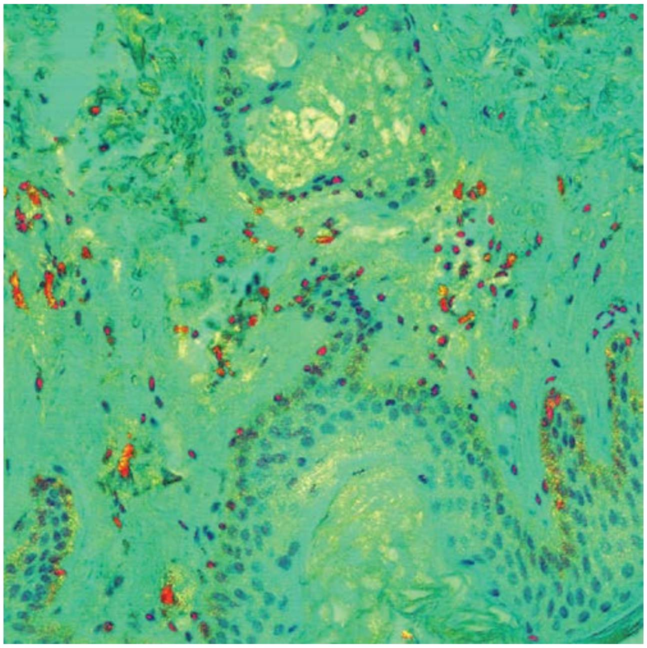

**Figure 36.30** Confocal laser microscope: Mast cell activation (red reaction) appears a very reliable parameter in distinguishing vital lesions (×200).

multifunctional cytokines, their potential contributions to the processes of inflammation have become increasingly evident. Histamine levels in skin vary appreciably after wounding, and the levels increase significantly between 5 minutes and 3 hours after trauma and decrease afterwards until 24 hours [4]. Furthermore, they appear early in vital response to trauma [5]. The evidence for MC activation appears a very reliable parameter in distinguishing vital and postmortem lesions (such as ligature marks) even when victims survived for a very short period and the time was insufficient for other reactions to develop and become reliable to distinguish from lesions of postmortem origin. Time-dependent changes of cytokine expression, which precede the inflammatory cells morphologically detectable, are very suitable markers of the early phase of wound healing, especially in cases of a short survival time [47] (Figure 36.30).

In conclusion, a complete immunohistochemical investigation of skin samples of ligature marks should be performed utilizing a wide panel of antibodies (anti-tryptase, fibronectin, TNF-α, IL-6, IL-8, IL-10, MCP-1, IL-15, IL-1ß, CD45, CD4, CD3, CD8, CD68, CD20, CD15), bearing in mind that tryptase, IL-15 and CD15 appear to be reliable parameters in the determination of the vitality of ligature marks with the accuracy needed for forensic purposes [48].

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# 37 Asphyxia due to Metabolic Poisons

*Henrik Druid*

Asphyxia is usually understood as a compromise of the oxygen delivery to the tissues. Several chemical agents may produce asphyxia in a wider sense via a variety of mechanisms. In this chapter, a selection of hazardous compounds that interfere with the cellular metabolism are treated. A number of additional agents are also included although their mechanisms of action are different.

## Carbon monoxide

During complete combustion of hydrocarbons, water and carbon dioxide are the final products, but in most situations, the oxygen concentration in the air is insufficient during a rapid onset, or consumed during a fire, resulting in the formation of carbon monoxide (CO). Upon inhalation, carbon monoxide binds approximately 220 times stronger to haemoglobin than oxygen to form carboxyhaemoglobin (COHb). A continuous breathing implies that COHb accumulates; hence the outcome depends on both the concentration of CO in the inhaled air and the time of exposure. Unfortunately, pure CO intoxication typically induces tachypnoea [2], speeding up the COHb formation. If the concentration is very high, unconsciousness, and even death, may occur within minutes. Since most modern motor vehicle engines today are equipped with catalysts, the number of suicides from inhalation of exhaust fumes in a car has dropped dramatically in most countries. A similar decrease of indoor suicides has also been recorded following the gradual shutdown of domestic gas supplies to many cities worldwide. As a consequence of the elimination of these sources, charcoal burning of briquettes in enclosed spaces started to increase around year 2000 as a means for suicide [23]. The effectiveness of small disposable barbecue grills for this purpose was spread over the internet, resulting in reports of such suicides in many countries. However, most fatal CO poisonings seen today are fire victims, and the vast majority of these are unintentional deaths [16].

COHb is a pigment with a bright cherry-red colour, which results in a brighter red colour of the venous blood, and hence to more or less pink postmortem staining of victims of CO poisonings (Figure 37.1). CO also binds to the ferrous iron of myoglobin, causing a brighter colour even to the skeletal muscles, which may be a helpful sign when a charred body is examined (Figure 37.2). Soot in the airways is, of course, a typical finding in deceased fire victims, and it indicates that the person was alive during the fire, but is not a proof of fatal carbon monoxide poisoning *per se*.

Due to the change of colour to the blood by increasing levels of COHb, spectrophotometric methods have been used for decades to determine the proportion of this form of haemoglobin [29] although accurate and more sensitive headspace gas chromatography–mass spectrometry (GC–MS) methods are also available today. These latter alternatives also offer better opportunities to analyze putrefied samples [13]. Small amounts (less than 0.5 per cent) of COHb may be detected in normal subjects due to the endogenous production of CO in the body, and heavy smokers can show up to 10 per cent COHb in their blood. Further, exposure to paint strippers containing dichloromethane is another source of elevated COHb levels that should not be overlooked. Dichloromethane is metabolized to CO in the body, and the COHb levels may continue to increase after interrupted exposure until the accumulated dichloromethane is eliminated. Although the hepatic conversion of dichloromethane to CO was discovered in the 1970s [24] and the toxicity of this solvent was reported as a workplace hazard, occasional fatalities are still encountered [19]. The toxicity is, however, mainly due to the central nervous system effects of dichloromethane itself and COHb elevations are moderate. In fire victims, levels exceeding 50 per cent saturation are usually seen in cases where carbon monoxide is considered to be solely responsible for the death, and somewhat higher percentages are seen in subjects who have inhaled vehicle exhaust fumes [16]. This discrepancy is assumed to be due to a contributory effect of other compounds in the smoke inhaled by fire victims [6].

Less well appreciated is that, in addition to its binding to haemoglobin and myoglobin, CO also inhibits mitochondrial cytochrome-c oxidase by binding to its haeme a3/$Cu_B$ site [7]. However, CO is a competitive inhibitor – i.e. competing with oxygen, whereas $H_2S$ and HCN are non-competitive inhibitors of this enzyme – and this mechanism is not considered to be of any major importance for the CO toxicity in practice.

In fatalities where circumstances suggest inhalation of exhaust fumes, but where the blood, livores and tissues do not show bright red colour, $CO_2$ intoxication might be considered. However, the possibility of methaemoglobin (metHb) formation should not be overlooked. A blood concentration of 56 per cent metHb was found in a man who had inhaled exhaust fumes in a car equipped with a catalytic converter; the concentration in the blood sample remained the same after 6 months of freeze storage [28].

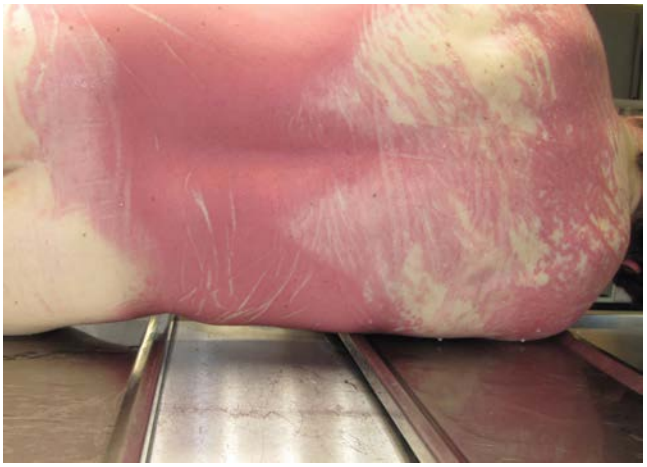

**Figure 37.1** Bright red livores of a man with COHb 71 per cent.

## Cyanide

Hydrogen cyanide is an extremely toxic gas (or liquid – hydrocyanic acid), which prevents tissue utilization of oxygen by binding to cytochrome oxidase, resulting in inhibition of cellular respiration. This binding occurs very rapidly and may cause death within minutes [22]. The expectation of a rapid death is probably a major reason for the ingestion of cyanide salts to commit suicide. However, the availability of hydrogen cyanide is limited and therefore such suicides are mostly seen in groups with ready access to it, such as scientists, jewellers and metal workers, a pattern that has been reported for decades and still remains unchanged [10]. Despite the awareness of its toxicity, workplace exposure causing non-intentional poisoning continues to contribute to the cyanide death toll. In addition, hydrogen cyanide can be formed in toxic concentrations in fires, and even if certain plastics have been banned because of a high production of cyanide upon combustion, many fire victims still show high blood cyanide levels. The high toxicity has also attracted interest by terrorists, and several attempts of mass killings have been reported, the most well-known being that in a Tokyo underground station in 1995, where bags of sulfuric acid and sodium cyanide were found in a restroom. When mixed, these would produce deadly hydrogen cyanide (HCN) gas.

Since the delivery of oxygen to the tissues is prevented, the oxyhaemoglobin concentration remains high and produces a characteristic cherry-red appearance of the livores of the victim. This colour change may not always be conspicuous. Darker postmortem staining, or duller hues, may be produced by cyanosis in cases with a somewhat prolonged agony, perhaps due to a paralysis of the respiratory muscles prior to death. A variable proportion of cyanmethaemoglobin formed, as well as the influence of environmental temperature on the oxygenation of haemoglobin after death, may also affect the colour of the blood, tissues and livores. Hence, if there is no suspicion of cyanide poisoning from the background information, the external examination of the body might in some cases raise this suspicion, whereas this possibility may be overlooked in other cases. A smell of bitter almonds from the gastric contents is another clue, but a proportion of the population is unable to detect this smell. Further, cyanide is corrosive, hence oral intakes may produce a reddening and oedema of the gastric mucosa.

It should be appreciated that not all cyanide poisonings are due to oral intake or inhalation of smoke in fires. There are a number of substances that are cyanogenic, producing

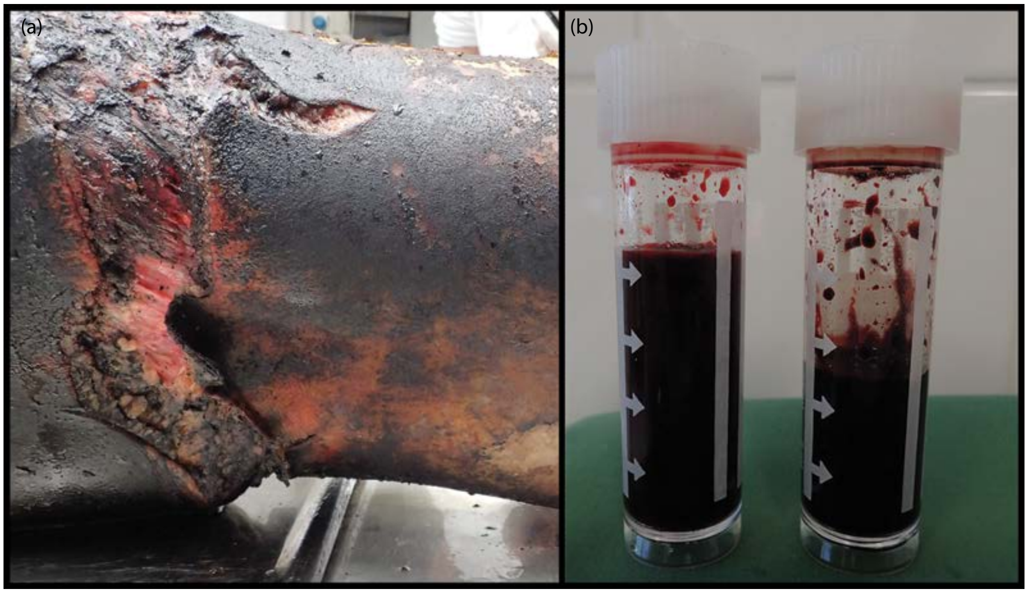

**Figure 37.2** (a) The back of a fire victim dying of carbon monoxide poisoning. Note the bright, pink colour of the exposed muscles, contrasting to the charred skin. (b) The femoral blood sample (left) compared to that of one collected from a hanging case (right).

cyanide after a variable time after exposure. These include the industrial solvent acetonitrile, which is biotransformed via cytochrome P450 to release cyanide [3].

## Tetrodotoxin

Tetrodotoxin is the toxin in puffer fish and can produce lethal poisoning at extremely low concentrations. It is also found in some other fish and aquatic animals such as certain species of octopus, newt and snail. Tetrodotoxin can also be produced by some bacteria including the *Pseudomonas* genus. The most prominent and serious effect is respiratory depression, which is due mainly to interference with the action potential generation in striated muscles, causing paralysis. Experimentally it was shown that, after i.v. tetrodotoxin administration, the phrenic nerve continued to elicit action potential for a substantial time after the diaphragm action potential stopped and diaphragm had ceased to contract [5]. Subsequent studies have shown that tetrodotoxin is a sodium channel blocker of excitable tissues (nerves and muscles) in mammals, whereas the animals producing this toxin are protected due to a substitution of the aromatic amino acid chain in the domain I of the sodium channels with non-aromatic amino acids [20].

Most poisonings reported regard ingestion of puffer fish, but a large outbreak of intoxications was reported in Thailand after ingestion of horseshoe crab eggs, involving 71 persons, one of whom died [17]. In three outbreaks of puffer fish poisoning in Bangladesh during 2008, 17 people died. Toxicological analysis of tetrodotoxin in blood in seven of the fatalities revealed concentrations >9 μg/ml [15]. Several liquid chromatography–mass spectrometry (LC–MS) methods have been described for sensitive selective detection of tetrodotoxin, however it should be kept in mind that there are a number of natural and synthetic tetrodotoxin analogues that also may produce severe poisoning [1]. There is no antidote, although artificial ventilation, started in the early phase, can prevent fatalities on most occasions.

## Opioids

For a very long time, opioid drugs have been the only alternative for the treatment of severe acute and chronic pain, and they remain so today. All opioid drugs share respiratory depressant effects, which limits their use. Further, due to their reinforcing effects, opioids are also widely abused. The consequence is that subjects exposed to opioid drugs are at risk for overdosing, and even though the majority of drug addicts are not using an opioid as their main drug, it is highly likely that, if they die of an acute intoxication, it will be an opioid.

Heroin is a semi-synthetic opioid, produced by acetylation of morphine extracted from opium poppies. This is a simple way to make the molecule more lipophilic, and hence more rapidly delivered to the brain, where it can exert its desired reinforcing effect. However, it is degraded first to 6-acetylmorphine and then to morphine, and it is believed that most of the effects are produced by morphine, even though it has been shown that the immediate euphoria most likely is conveyed by 6-acetylmorphine since the levels of this intermediate correlates with the dopamine release in micro dialysates from striatum [11]. The degradation is rapid, implying that, in most deaths due to acute heroin intoxication, morphine will be detected along with a very low concentration of 6-acetylmorphine. However, about 40–50 per cent of deaths due to opioids, including heroin, are delayed, ensuing after an hour or more of hypoventilation, and during this time the 6-acetylmorphine will be completely eliminated. Analysis of the urine for 6-acetylmorphine is therefore recommended in order to confirm an intake of heroin rather than morphine. This distinction is important since the blood concentrations of morphine in acute heroin intoxications are perhaps 10 to 100 times lower than those causing death from intake of morphine tablets. Further, microscopic examination of the brain for hypoxic cell injury of Purkinje cells in the cerebellum and CA2 neurones in the hippocampus is particularly important to support the diagnosis of delayed opioid toxicity death.

The toxicity of heroin and other opioids is highly dependent on the degree of tolerance. Patients with severe pain as well as drug addicts will need to increase the dose over time to achieve the analgesic and euphoric effect, respectively. This tolerance is pharmacodynamic, implying that the blood concentrations will be quite different, as opposed to the tolerance induced by most other drugs, which is pharmacokinetic, involving increases in the metabolism and excretion. Opioid overdose death is a misnomer, since the drug users most likely took a regular dose, but misjudged their degree of tolerance. In acute heroin toxicity deaths, the blood morphine concentration is often reported to average 0.3 μg/ml, but with a wide variation. This great variation is true also for most other strong opioids.

The classical sign of an opioid toxicity death, regardless of substance involved, is a massive lung oedema and foam in the airways, sometimes protruding from the mouth and nostrils (Figure 37.3).

The mechanism behind opioid toxicity death is incompletely understood. However, intensive care patients treated with morphine for severe burns can show much higher blood morphine concentrations than those dying from morphine overdose, but they probably would die too if they were disconnected from the respirator. Hence, respiratory depression is believed to be the main reason for opioid deaths. This has been shown experimentally to be due to an overstimulation of mu opioid receptors on the interneurones in the respiratory control centre (pre-Bötzinger complex, pBC) in the medulla oblongata,

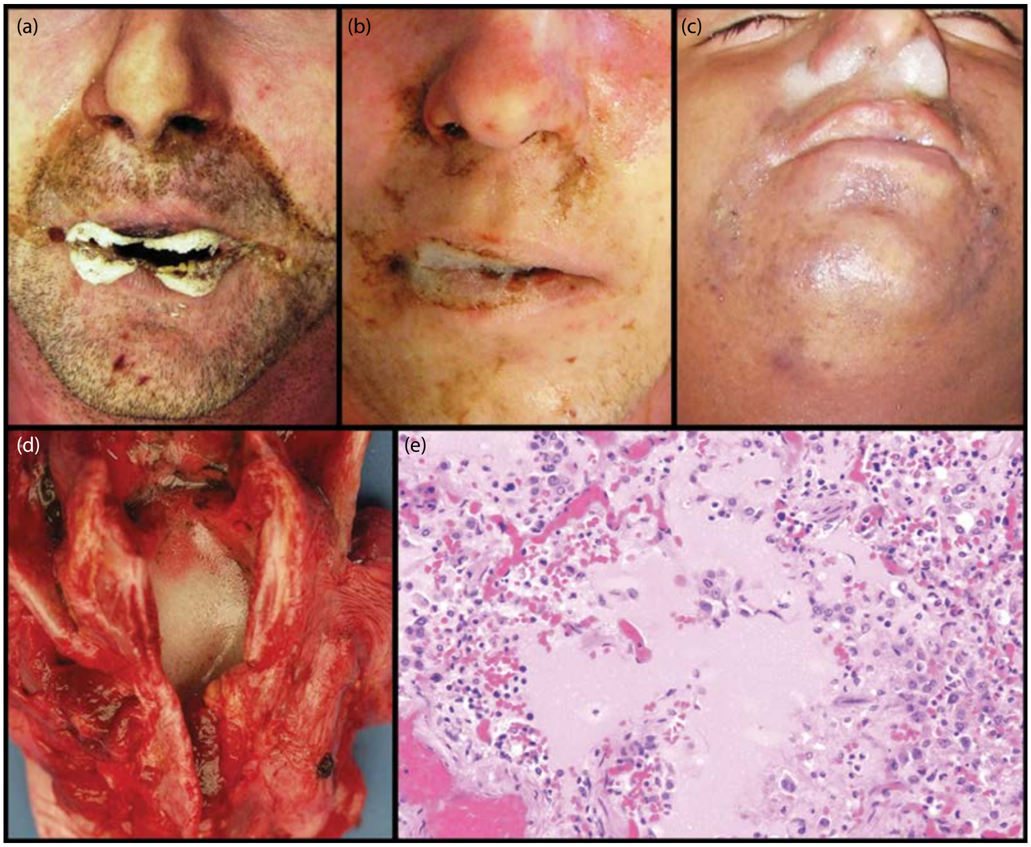

**Figure 37.3** (a)–(c) Three opioid toxicity deaths after heroin, methadone and buprenorphine administration. (d) If removed from the mouth and nose, froth may be seen in the lower airways. (e) Massive lung oedema is often conspicuous. H&E staining. Original magnification 200x. These findings are typical of most deaths caused by strong opioids.

preventing them from signalling to the motoneurones in the cervical medulla responsible for the diaphragm operation. The human counterpart is located in the ventrolateral medulla, but despite developments in immunohistochemistry, cell-signaling analysis and autoradiography, there is today no method that can be used to determine if the pBC interneurones have been overstimulated and thereby to obtain support for an opioid overdose. And, to reiterate, the blood drug concentrations are not helpful either because of their extreme variation with the degree of tolerance.

In the absence of a marker of opioid tolerance, hair analysis may be used as a proxy. In two studies the hair concentrations of morphine in fatal heroin intoxications were reported to be lower in heroin overdose death than in street heroin users [8,26]. Careful segmental hair analysis can map the recent and past exposures and serve as a guidance for the interpretation in obscure cases [9]. In the vast majority of cases acute heroin toxicity deaths were found to have used no opioids during the preceding 2 weeks (unpublished observations), implying an increase in sensitivity for the respiratory depressant effect of a new exposure.

In recent years, new psychoactive substances have been introduced via internet sites. Whereas most of these are psychostimulants or hallucinogenic, a few may be opioid agonists. One of these, MT-45, was apparently not recognized as an opioid by all users, resulting in many deaths until it was classified and disappeared from the market [21]. In recent years, fentanyl analogues have been produced on a large scale and many of these can be purchased as nasal sprays from internet sites. Acetylfentanyl was among the first to appear on the market and caused a large number of deaths [25,30]. Furanylfentanyl appeared on the market for a short period, and when found in postmortem cases, it was almost always in the context of an opioid overdose [12]. Hence, the mere detection of a fentanyl analogue should raise the suspicion of an opioid toxicity death. The increase in the fentanyl deaths, and in deaths from other opioids has been so great that US President Donald Trump declared the opioid crisis a public health emergency in October 2017.

## Other asphyxiants

There are a number of other substances which may cause asphyxia. Azides are present in airbags, primers, biocides and laboratory reagents. Hydrazoic acid/hydrogen azide ($HN_3$) is formed when sodium azide comes into contact with sulfuric acid. Severe poisonings may be caused by industrial exposures, and suicide attempts have been reported among laboratory workers [4].

Strychnine is a classic poison which eliminates the inhibition of all nerves and results in tetanus-like convulsions. Deaths are generally due to asphyxiation caused by failure of the respiratory muscles to contract in a regular pattern [27].

Asphyxiants are rare among pharmaceutical drugs. However, amobarbital, which was used in the past as a tranquilizer and hypnotic, selectively inhibits the oxidation of NADH-linked substrates and thereby inhibits the mitochondrial electron transport chain [14]. This drug is no longer marketed, but barbiturate intoxications with preparations that have been removed from the market still occur since many people keep drugs for long periods of time.

Organophosphorus compounds present in many pesticides are responsible for a substantial number of deaths, particularly in the least developed, low-income countries, and are known to produce respiratory arrest. Although acetylcholinesterase inhibition is believed to be the pathophysiological mechanism behind the respiratory failure, it has recently been observed that a hypoperfusion of the respiratory control centres in the brainstem contributes to the inhibition of the respiratory muscles [18].

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# 38 Survived Neck Compression

*Stefan Pollak and Annette Thierauf-Emberger*

Survived injuries from manual and/or ligature strangulation are a common issue in clinical forensic medicine, whereas abortive attempts of hanging are rare. In this chapter, some aspects of survived strangulation are discussed.

## Manual and ligature strangulation

In neck compressions by an offender, most victims are female, whereas the perpetrators are mainly males [15]. Assaults to the neck often occur in sexual offences, fights or robberies [33]. Statistically, the number of manual strangulations is higher than that of ligature strangulations. In a large percentage of survived strangulations, both the offender and the victim are intoxicated [12]. The perpetrator is often somebody close to the victim (husband, life partner, ex-boyfriend, neighbour, colleague, etc.) [12].

Local injuries on the neck range from vague reddening via different forms of abrasion to intra- and subcutaneous haematomas. Redness of the skin is mostly patch- or streak-like (Figure 38.1). It is often associated with superficial epidermal defects and intracutaneous bleeding. Mere reddening of the skin remains visible for about 2 days after the incident at the most [20]. Consequently, victims of assaults to the neck should be examined as soon as possible. The same applies to suspects, as strangling perpetrators may be injured themselves when they meet resistance. Scratches from fingernails are particularly frequently seen on the offender's face and neck.

Skin marks from manual strangulation include roundish, oval or confluent bruises caused by pressure of the finger tips (Figure 38.2) as well as scratch-like and crescent-shaped abrasions from the fingernails. External findings on the neck may be insignificant or even completely absent, especially if the pressure was exercised over a large area (Figure 38.3) or soft objects such as a cushion or scarf were interposed [12].

In ligature strangulation, the external findings are strongly dependent on the properties of the tool used, the intensity and duration of strangulation and the victim's resistance. Usually the ligature mark more or less encircles the neck, presenting as streak-like reddening, which is sometimes accompanied by excoriations [29]. Strangulation tools may show not only skin flakes from the victim but also DNA traces from the perpetrator due to them powerfully pulling on the ends of the ligature [4].

Figures on the frequency of visible neck lesions vary. Some victims do not show any visible signs of the assault, but report pain in the laryngeal region or swallowing problems.

Typical symptoms after neck compression are dysphagia, dysphonia, tenderness and pain of the laryngeal region, especially when moving the neck. The percentage of victims losing consciousness during the assault varies depending on the composition of the study material. The same applies to the frequency of involuntary loss of urine and faeces, which almost exclusively happens in association with unconsciousness. Retrograde amnesia was reported in a quarter of the victims who had become unconscious in the course of neck compression [33].

Where internal injuries are suspected, a supplementary radiological and ENT examination should be performed [38]. Occasionally, fractures of the cricoid cartilage and tracheal disruption can lead to stridor and life-threatening respiratory distress. If the space between the larynx and the spine is infiltrated with blood or oedema fluid, the normal laryngeal crepitus felt in movements from side to side may be absent [11].

Blood extravasations due to congestion provide important diagnostic clues not only in cases of fatal strangulation, but also in survived assaults to the neck. It has to be emphasized, however, that this finding is not a specific sign of asphyxia. Petechial haemorrhages in the conjunctivae have been repeatedly reported in physiological processes involving a pressing effect (labour, fits of coughing, massive vomiting) [10]. In surviving strangulation victims, the presence of petechial bleedings depends on several factors, i.e. duration, intensity and kind of neck compression as well as the relative strength of the persons involved. Isolated petechiae in the eyelids and conjunctivae may escape detection after only a relatively short period of time. On the other hand, there are examples in which it took several weeks until a confluent subconjunctival haemorrhage (hyposphagma) disappeared completely.

Petechiae of the facial skin, the conjunctivae and the other mucous membranes of the head and neck found after prolonged strangulation (see Figures 38.1 and 38.3) are caused by an increase in intravascular pressure and do not indicate a lack of oxygen. Obstruction of venous outflow and at least partly maintained arterial inflow (vertebral arteries) are considered essential causes of congestion. In strangulation using the forearm, obstruction of arterial blood flow to the brain is the major pathophysiological mechanism resulting in a rapid loss of consciousness.

The potentially life-threatening effects of neck compression are essentially due to occlusion of the cervical

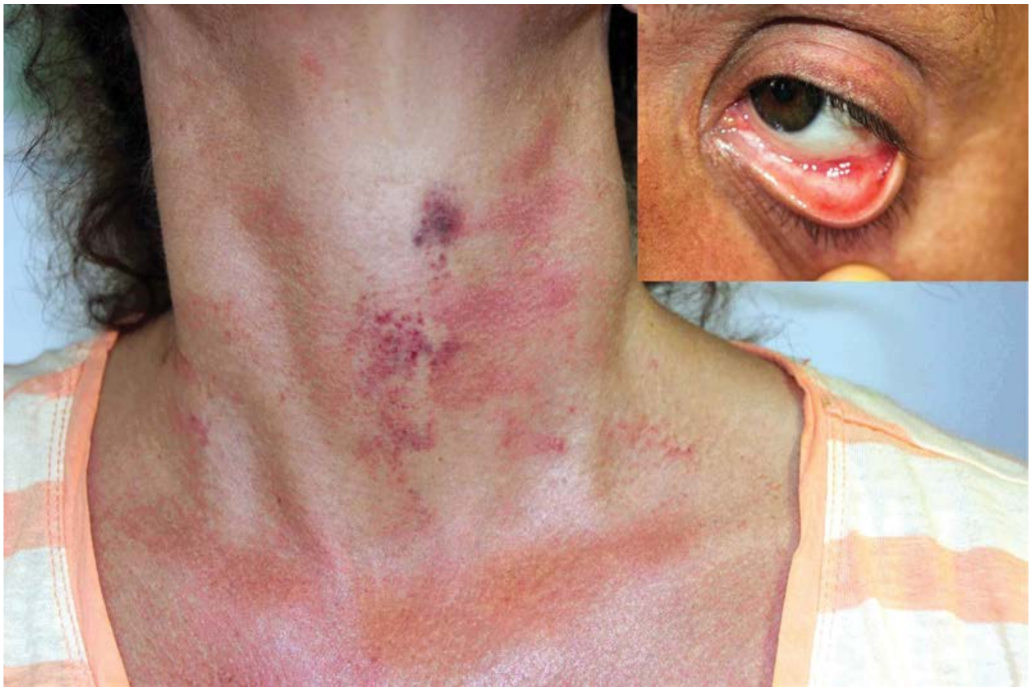

**Figure 38.1** Front of the neck (46-year-old woman) with multiple erythematous markings as well as intra- and subcutaneous haematomas. The victim was strangled with both hands in a supine position and lost consciousness. Medicolegal examination and photo-documentation took place about 5 hours after the offence. Note the moderate subconjunctival haemorrhage (box in the right upper corner).

vessels and/or obstruction of the airways [13]. The role of vegetative reflex mechanisms is debated. In the more recent literature, many authors are of the opinion that sudden death due to a short grip to the neck is possible only in individuals with a respective disposition and does not occur under normal circumstances [19]. In this context it is interesting that articles dealing with sports medicine do not report on carotid sinus reflex deaths, although considerable pressure is applied to the lateral cervical region in some forms of martial art [24].

Apart from local findings on the neck and signs of venous congestion [28], a high percentage of strangulation victims also show concomitant injuries in other body regions. These are mostly caused by blunt traumatization, such as blows to the face (Figure 38.4) or defence injuries on the upper extremities.

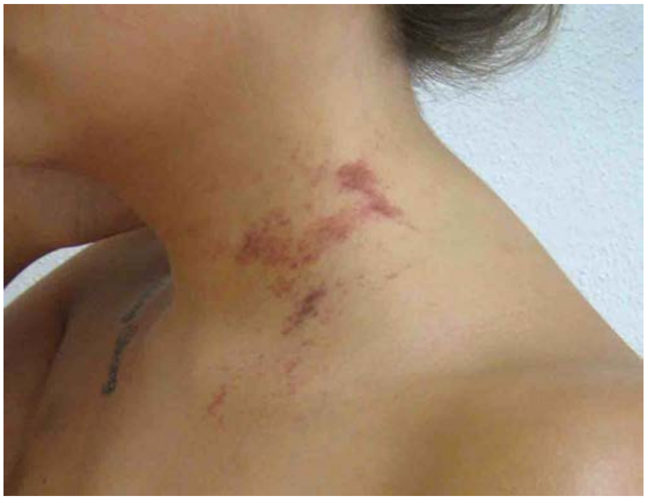

**Figure 38.2** Left side of the neck with multiple and partly confluent haematomas from manual strangulation. The victim, a 22-year-old woman, was throttled until she lost consciousness. The photo was taken 36 hours after the incident.

## Survived attempts of hanging

Cases of survived hanging are rare in forensic medicine. As these are predominantly suicide attempts, which are not subject to punishment as such, no forensic investigation is ordered in most instances. This may explain why the number of reports in the medicolegal literature dealing with findings after failed attempts of hanging is relatively small (e.g. [5,9,26,30]). In contrast, numerous articles on the epidemiology, course and outcome parameters of survived hanging attempts can be found in the clinical literature (e.g. [1–3,6–8,14,17,18,22,23,27,31,35,37]).

Some situations require detailed investigation of the facts and circumstances, for example where it is unclear whether the event was a suicide attempt or homicidal manual/ligature strangulation with subsequent simulation of self-hanging. When interpreting the pattern of findings, one has to pay attention to injuries caused later during recovery and resuscitation. Occasionally, a suicidal act

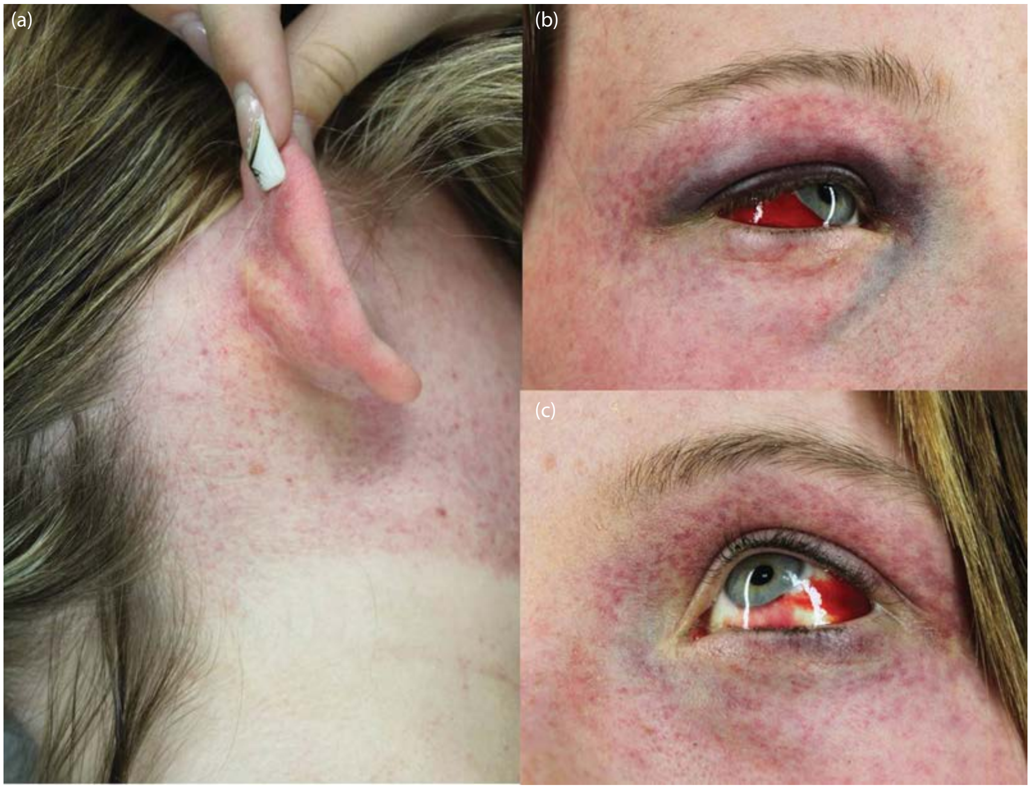

**Figure 38.3** (a) Right auricular and lateral neck region of an 18-year-old woman who was throttled by her boyfriend in the course of an argument until she lost consciousness. Pressure on the neck was exercised with the flat of both hands placed such that the thumbs were located in front of the larynx. Note the absence of definable grip marks. As a consequence of blood congestion, the facial skin is densely dotted with petechiae above the strangulation level. (b, c) Subconjunctival haemorrhage (hyposphagma) is covering the sclera of both eyeballs. The interval between neck compression and photographic documentation was 2 days.

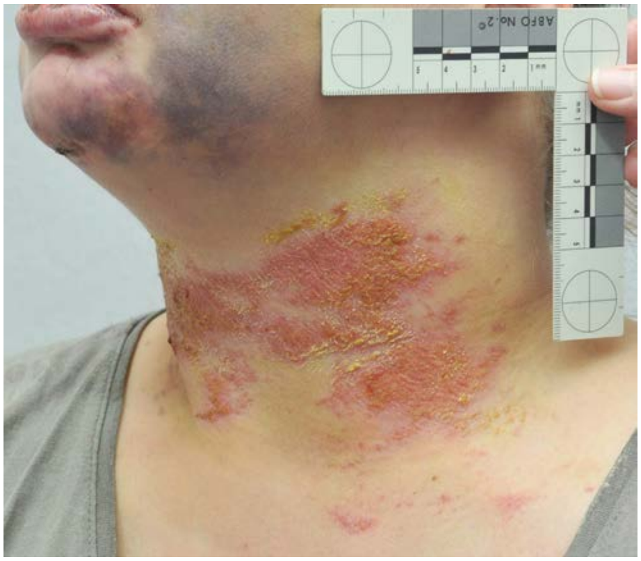

**Figure 38.4** Left mandibular and neck region of a 26-year-old woman who was manually strangled 2.5 days before. There are extensive abrasions of the neck skin covered with scab. The concomitant haematomas have already assumed a yellowish colour in their marginal parts. Confluent haematomas on the face were caused by multiple fist blows. The examination also revealed bilateral hyposphagma and a fractured hyoid.

may be fatal even if the planned strangulation failed but an unforeseen secondary trauma occurs, such as a head injury due to a fall or drowning ('complicated suicide' [34]).

With regard to the clinical course, most victims of survived hanging attempts are in a state of deep coma at first. They may also show cerebral seizures, and even status epilepticus induced by the hanging has been observed [21,27]. Analogous symptoms have been reported following a prolonged choke hold [32]. In the post-comatose stage, brain dysfunction manifests as cognitive and motor deficits as well as memory and attention disorders [1], which may persist for a long period of time [7]. Only in exceptional cases is memory access to the events immediately before hanging maintained.

Where there is initial cardiac arrest (CA), severe neurological sequelae have to be expected in a high percentage of patients [6]. Conversely, non-CA patients usually have a good neurological outcome [8,14,36]. Hanging time, presence of cardiopulmonary arrest at the scene and Glasgow Coma Score (GCS) on admission are the most important prognostic factors for the clinical outcome [22,23]. Among those who survive the hanging attempt, the probability of complete neurological recovery is high [18].

Apart from the consequences of global hypoxia such as cerebral oedema and multifocal encephalomalacia

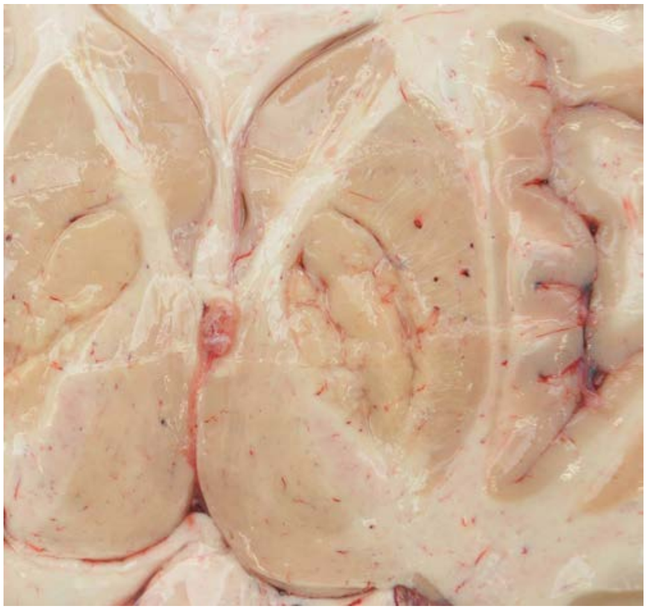

**Figure 38.5** Basal ganglia (horizontal section) showing bilateral cerebral softening (encephalomalacia) of the globus pallidus (medial part of the lentiform nucleus) in a case of attempted hanging using a towel (31-year-old man, survival time 5 days in a permanently comatose state).

(Figure 38.5), defined cerebral lesions may also occur. Two potential mechanisms are to be considered:

- Thrombotic or embolic vascular occlusion [5].
- Hypoxic brain injury triggered by strangulation in cases of pre-existing arteriosclerosis.

Thromboembolic complications most frequently develop if the carotid artery intima has torn due to overstretching. These lesions are typically localized just below the carotid bifurcation. The neck vessels may also show wall dissection with intramural haematoma [17]. Carotid dissection can occur in survived manual strangulations [21].

Fractures of the cervical spine are rarely seen in survived attempts of hanging [2,3,17,35]. The frequency of injuries to the hyoid bone and laryngeal cartilages depends on the modalities of hanging (freely suspended or supported), the course of the noose, and the age and sex of the victim. If there is pulmonary oedema [6,25,36], foam may emanate from the mouth and nose.

The morphology of the hanging mark is extremely variable. This applies not only to lethal acts of hanging but also to survived attempts. Essential factors are:

- Width and surface properties of the ligature.
- Tensile force acting on the noose.
- Duration of suspension.
- Abrading relative movement between the ligature and the skin (as the noose tightens).
- Time interval between suspension and examination.

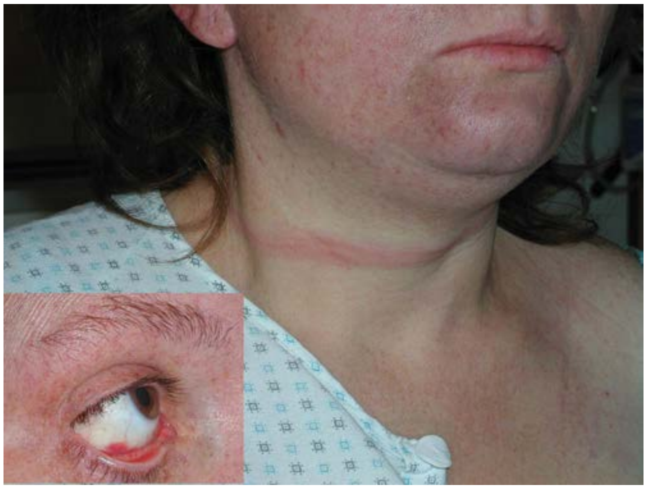

**Figure 38.6** Survived suicide attempt by hanging (39-year-old woman). Medicolegal examination and photo-documentation of the relevant findings took place 6 hours after rescuing the victim by removing the noose (single loop formed by an electric cable attached to the balcony wall). Reddish, streak-like hanging mark, pronounced signs of blood congestion with petechial haemorrhages in the facial skin and hyposphagma (box in the left lower corner) can be seen.

In surviving victims, subcutaneous bleeding may occur under the hanging mark [5,9,29], while in primarily fatal acts of hanging such bleeding is prevented by persistent compression of the tissue. A permanent skin impression (furrow), as is often seen in hanged persons with a prolonged suspension time, is not to be expected in surviving victims (Figure 38.6). The hanging mark is level with the surrounding skin or even protrudes from it (in the presence of tissue oedema and/or haematoma).

Both lethal acts and survived attempts of hanging can be associated with blood congestion above the strangulation level. Pathophysiological conditions for the formation of a congestion syndrome are occlusion of the cervical veins with at least partial patency of the carotid and/or vertebral arteries. This constellation is more probable if the body is substantially supported (e.g. in a crouching position) and if the course of the noose is atypical. In survivors, the conjunctival petechiae tend to form a confluent haematoma (hyposphagma), which may remain visible for several days or even weeks.

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# 39

# Pitfalls and Mistakes

*Stefan Pollak and Annette Thierauf-Emberger*

This chapter considers the potential sources of error in the assessment of death from suffocation and pressure on the neck. An exhaustive enumeration of all misleading findings and consequent misinterpretation is, of course, not achievable. As in many other fields of practical casework, mere knowledge of the pertinent literature cannot compensate for a lack of experience on the part of the examiner. This insight was clearly expressed around 300 years ago by Giovanni Battista Morgani (1682–1771) who is quoted as follows at the beginning of *Knight's Forensic Pathology* [17]:

> 'Those who have dissected or inspected many bodies have at least learned to doubt, while those who are ignorant of anatomy and do not take the trouble to attend to it, are in no doubt at all.'

The following discussion should therefore be understood as an invitation to scrutinize the diagnostic value of signs allegedly militating for or against a certain type of mechanical 'asphyxia'. According to the main causes of error, the case examples compiled in this chapter are classed in three categories:

- Minor manifestation or absence of indicative clues.
- Spurious signs seemingly pointing to death by suffocation or pressure on the neck.
- Misleading findings and evidence hard to explain.

## Minor manifestation or absence of indicative clues

Especially in infants and very old people, but also in care recipients of all age groups, *smothering* does not necessarily go along with external damage to the facial skin. It is a matter of common knowledge that a considerable number of persons incapable of resistance have been killed but were thought to have died from a non-violent cause such as sudden infant death syndrome (SIDS) or cardiovascular disease. The risk of misjudgement is not confined to the external examination of the corpse only. Even a complete forensic autopsy does not ensure that homicides committed by smothering are detected. In some cases, the crime is elucidated only due to a later confession of the perpetrator [2,12]. Blockage of the nostrils and mouth may be induced by hand, a soft fabric or by pressing a cushion on the face. Other kinds of inconspicuous suffocation include a plastic bag put over the head [6] or cling film (plastic wrap) covering the respiratory openings until breathing ceases [2]. As far as infants are concerned, pulmonary haemorrhages in association with acute emphysema of the lungs may raise suspicion since these histological findings are atypical for SIDS. In older victims, the haemorrhagic-dysoric syndrome has been characterized as a morphologic correlate of mechanical asphyxiation [3].

The autopsy appearance in *manual strangulation* is widely heteromorphic. Apart from clear cases with marked bruising and abrasion on the neck, the external (and sometimes also internal) evidence may be extremely scanty. Possible reasons include pressure exerted with the palm of the hand (Figure 39.1a), the forearm or the crook of the arm over a large area [9,19], but also the interposition of a soft fabric between the strangling hand and the neck as well as the grip of an assailant with bitten nails [5]. If the neck belongs to the dependent surfaces of the body, postmortem hypostasis sometimes makes it difficult to distinguish any additional bruises.

It is the authors' experience that some forensic pathologists fail to examine the neck structures in their entirety up to the mandible where the bony support facilitates the occurrence of pressure-induced contusions. Fingernail marks and scratches are often inconspicuous shortly after infliction and only become visible as a result of drying. It is therefore not uncommon that abrasions on the neck are overlooked during the first inspection, but easily diagnosed when re-examining the corpse at a later date. For that reason, it has been recommended to perform a second external examination of the body, at least in critical cases [8]. Especially in victims recovered from water, it takes some time until the moistened abrasions assume a brownish colour due to drying [19].

In fatal *strangulations by ligature*, one might expect at least one mark roughly corresponding with the width of the device applied to constrict the neck. However, frequently the mark is seen on only a part of the neck circumference rather than completely encircling it. If there is a concomitant haematoma in the underlying corium and/or subcutis, the resulting skin discolouration is usually faint. Abrasions often become clearly visible only after drying has produced a brownish discolouration. In suicides and accidental strangulations (e.g. due to autoerotic practices), the ligature usually remains in position for a prolonged period of time. Consequently, the long-acting pressure on the neck may leave a deeply embedded groove. In other cases, the local findings are restricted to a band-shaped pale area without

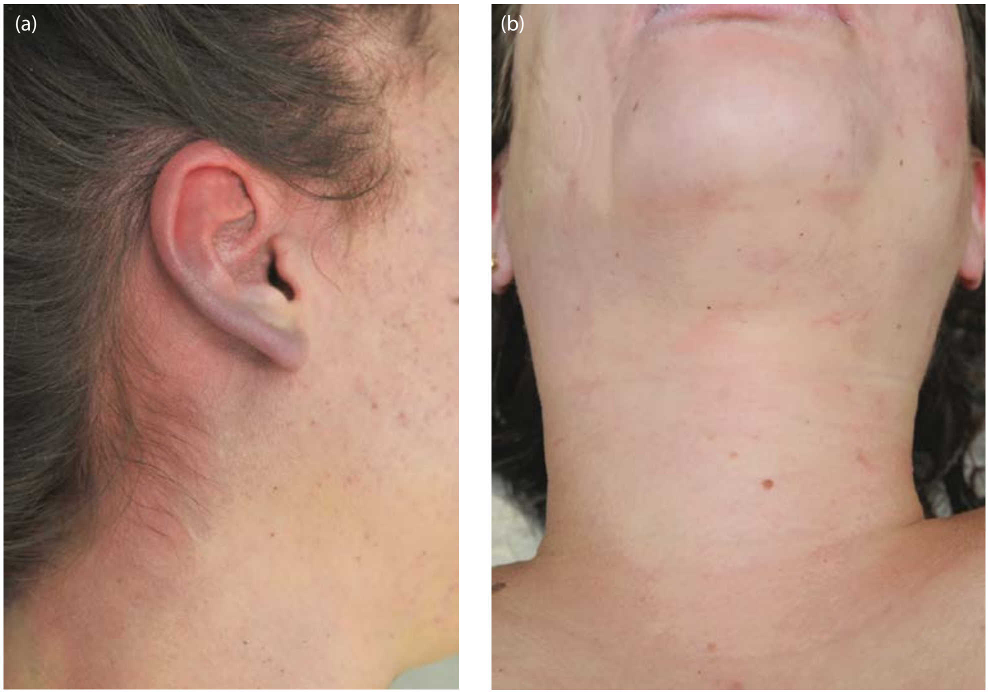

**Figure 39.1** Absence of external signs militating for strangulation. (a) Right lateral neck region of a woman killed by neck compression exerted with the flat of the hand. (b) Anterior neck region of a woman killed by ligature strangulation with a broad woollen scarf and subsequent drowning of the unconscious victim.

hypostasis (Figure 39.2). In some instances, the external appearance of the neck is not at all indicative of preceding ligature strangulation. This applies particularly to victims whose ligature is removed either by the perpetrator or by another person arriving at the scene before the investigators. The absence of significant skin marks is characteristic of nooses consisting of soft and wide fabrics such as scarves and towels (Figure 39.1b).

In *hanging deaths*, the local findings on the neck skin are subject to similar factors as discussed in the context of manual and ligature strangulation. In some circumstances, the hanging mark may be quite unremarkable. It is therefore not surprising that deaths by hanging may even be overlooked by the medical examiner [11,19], particularly if the body is no longer suspended and the noose has been removed. A vague hanging mark can be seen in victims suspended only for a short time and/or with the body being supported (e.g. in a sitting, kneeling or lying position) so that only a small part of the weight tightens the noose. Broad hanging devices made of soft fabrics and cushioned nooses also produce marks which are less impressive than those caused by rough ropes and cords.

Considering the great variability of the external findings in deaths from smothering and strangulation, a holistic approach seems recommendable when examining relevant cases. This means taking into account the individual background, the situation at the scene, the clothing, any concomitant injuries, any signs of congestion and, of course, the autopsy results.

## Spurious signs seemingly pointing to death by suffocation or pressure on the neck

Some manifestations of *postmortem changes* may lead to the assumption of death from mechanical ‘asphyxia’. In the early hours after death, the initial stage of hypostasis often forms blotchy purplish discolourations resembling grip marks if located on the neck. In most cases, the colour of hypostasis is originally bluish due to the lack of oxyhaemoglobin. Therefore, postmortem lividity of the face and neck can be confused with cyanosis induced by premortem blood congestion. Especially in prone and head-down positions, hypostatic skin haemorrhages occur within the dependent areas, sometimes simulating petechial haemorrhages as seen in the face and eyes after neck compression (Figure 39.3a).

In the presence of a deep neck fold, postmortem lividity is excluded by local pressure so that a pale linear stripe appears when the skin is stretched. The distinct colour contrast may imitate a ligature or hanging mark (Figure 39.3b). A deep horizontal neck crease can be suggestive of an indentation from ligature strangulation, especially after refrigeration of the body, implying congealing of the subcutaneous fat [20] (Figure 39.3c). If a person loses consciousness and remains lying in prone position beyond death with the anterior neck region being supported by a hard object, the contact area tends to become brown and leathery as though it had been abraded in an assault [15].

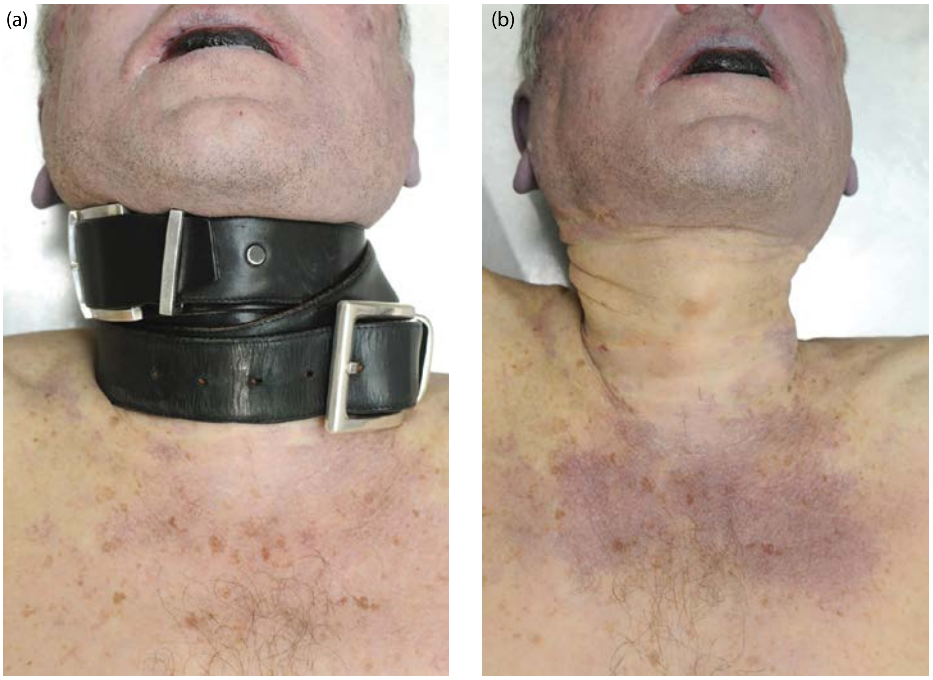

**Figure 39.2** (a) Fatal sexual asphyxia from neck constriction by two leather belts. (b) Note the sparse findings after removal of the ligatures. The neck skin appears pale compared with the face and the upper chest area.

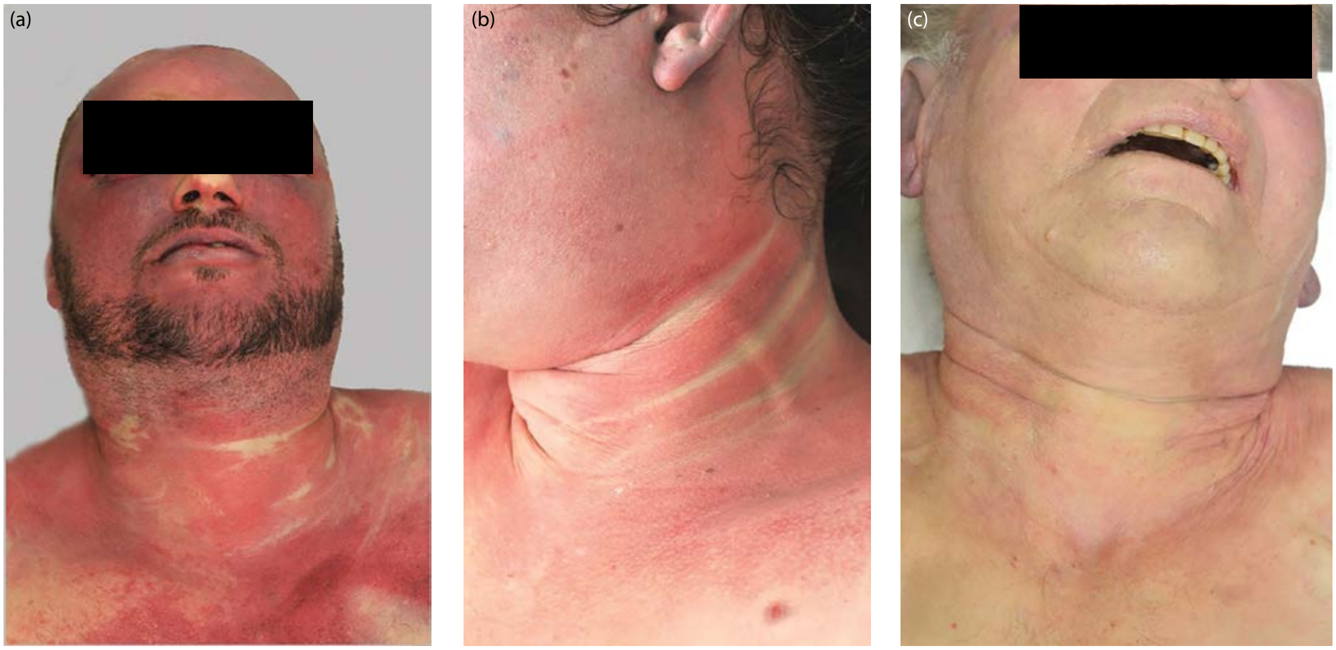
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**Figure 39.3** (a) Hypostasis in the facial and neck regions simulating cyanosis in combination with petechial haemorrhages in the conjunctivae. Death from dilated cardiomyopathy. The body was lying in prone position for several hours. (b) Hypostasis in the left lateral neck region, interrupted by blanched stripes from skin folds. (c) Deep neck creases similar to indentations from ligature strangulation.

Putrefactive changes such as marbling are easily mistaken for haematomas from manual strangulation. Skin-slippage with consecutive detachment of the epidermis and burst blisters are followed by exsiccation of the denuded corium, which assumes a parchment-like appearance similar to abrasions. Postmortem artefacts from ant bites can mimic premortem excoriations suggesting an assault to the neck [4,10]. In advanced putrefaction, tissue swelling by gases causes a circular groove on the neck if a shirt with a tight collar is worn [19].

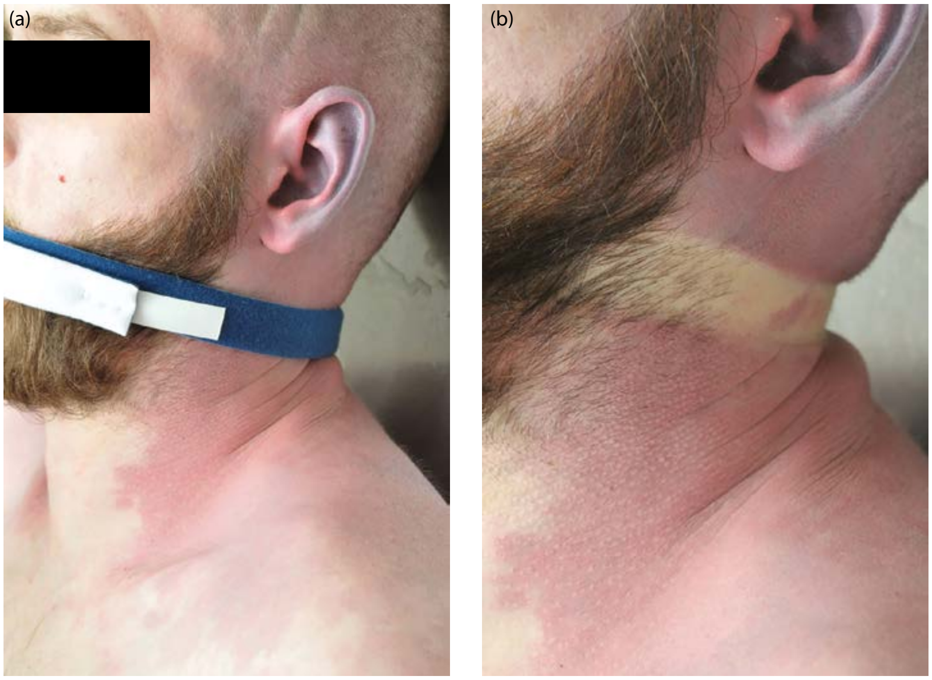

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**Figure 39.4** (a) Briefly survived drug intoxication. (b) The fastener of the endotracheal tube has left a pale streak within the hypostatic skin discolouration.

Chin straps are often attached on corpses by undertakers who want to keep the deceased's mouth closed until postmortem rigidity stiffens the masticatory muscles. The furrow left after removal of the strap may resemble a hanging mark passing around the neck and rising upwards to the occipital region. In dressed bodies, livor can be blanched under a tight shirt collar, causing a circular pale mark. Similar findings are produced by cervical collars used to support a patient's neck and by ribbon-like fasteners of oral endotracheal tubes (Figure 39.4).

Attempts at resuscitation including mouth-to-mouth insufflation sometimes entail abrasions around mouth and nostrils that may give rise to the suspicion of smothering. Most of these skin lesions are nail impression marks located on the lower face and in the submandibular regions [7]. Catheterization of the internal jugular vein is typically accompanied by perivascular blood extravasation which might be mistaken for a haematoma caused by blunt force. Improper rescue or transportation of a body by gripping the neck or using a noose might also suggest strangulation [22]. If a deceased is shaved before inspection of the corpse, parts of the corneal layer can be detached with consecutive parching of the neck skin possibly imitating strangulation marks.

Finally, the importance of careful *dissection* must be stressed. In order to avoid artefacts from postmortem blood extravasation, prior removal of the heart and brain is recommended [18,19]. This technique drains the neck veins of blood and thus reduces the risk of dissection artefacts [5]. However, there is no doubt that, in some cases, postmortem haemorrhages can already be present in the subcutaneous tissue and the muscles of the neck before an autopsy is performed. This particularly applies to bodies in a prone and/or head-down position. Prinsloo and Gordon [14] described artefactual postmortem haemorrhage between the oesophagus and cervical spine. Another artefact found in the neck tissues is 'banding' of the oesophagus [17], characterized by a pale area due to local absence of inner hypostasis. In cases of drowning, intramuscular haemorrhages of the neck muscles occur irrespective of any premortem strangulation [16]. These extravasations have been attributed to agonal convulsions, hypercontraction and overexertion of the affected muscle groups.

Incorrect handling of the body during either transport or dissection can cause postmortem fractures of the laryngeal horns [17]. There are a great number of anatomy variants of both the hyoid bone and the thyroid cartilage, which may be misinterpreted as alleged signs of neck compression. When the body is positioned for removing the neck structures during autopsy, a block is usually placed under the shoulders. This procedure is inclined to cause a subluxation of the lower cervical spine ('undertaker's fracture' [17]).

## Misleading findings and evidence hard to explain

This chapter deals with unusual constellations of findings which might be confusing at first glance. As a matter of course, the examples given here do not represent the whole spectrum of eventualities seen in forensic practice.

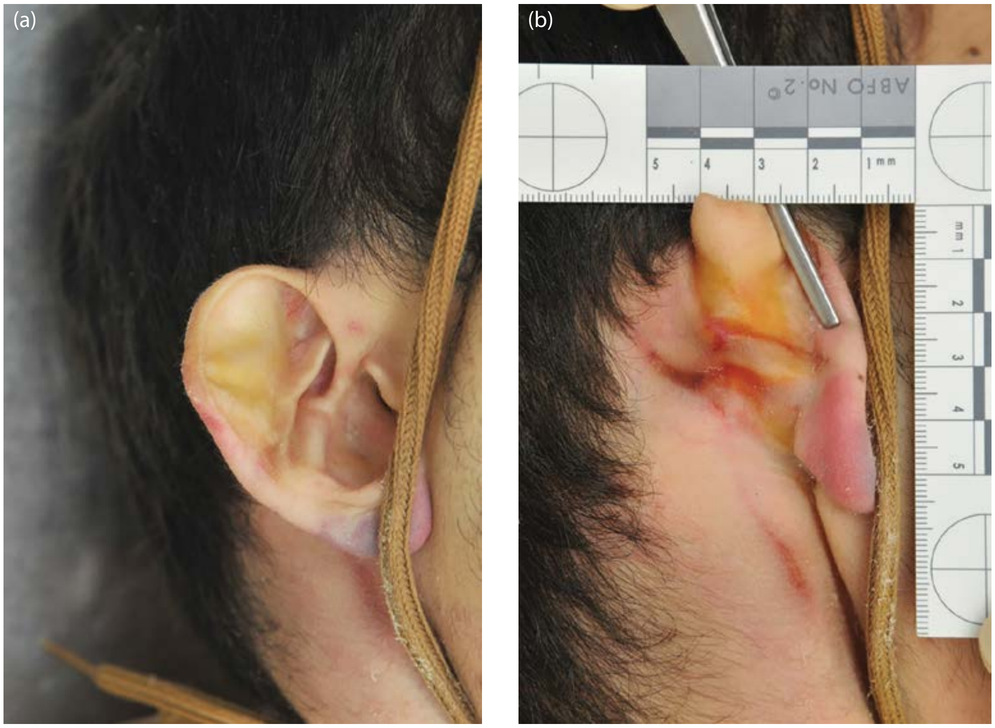

**Figure 39.5** Hanging victim suspended on a shoelace, which first ascended behind the ears but secondarily slipped to its final position in front of the ears (a). (b) Note the brownish-red streaks on the medial surface of the auricle.

In hanging deaths, fingers can get wedged between the loop and neck. As a consequence, the hanging mark is interrupted by parallel, vertical or oblique skin impressions as wide as a finger with ridged strips in between [13]. In some instances, the hanging device tightens step by step during suspension until the noose reaches its final position [19]. The successive upward movement of the ligature may produce a second [19] or a broadened mark seemingly conflicting with the width of the hanging device. The sliding noose may leave corresponding abrasions not only on the neck but also in the face and even on the medial surfaces of the auricles (Figure 39.5).

The presence of two hanging marks is usually attributable to the application of a noose with two ligature turns. An abortive attempt of hanging followed by a second try is an alternative. Possible reasons why an attempted suspension fails the first time are manifold, including a broken rope, suspension from a snapping branch or early rescue with regained ability to act.

Some victims of hanging may show blunt injuries suggesting infliction by an offender. In hanging deaths without third-party involvement, blunt traumatization can occur both in the premortem and the postmortem period. If a person intending suicide falls to the ground (e.g. from an elevated support before the loop is placed around the neck, or owing to a broken rope), respective injuries to the head or other body regions are to be expected [19]. Scalp lacerations in particular are frequent sources of external haemorrhage leaving suspicious blood stains at the scene (Figure 39.6). After death, a suspended body is at risk of striking the floor when cut down by relatives or other first aiders.

It is common knowledge that decapitation from suicidal hanging happens if the body falls from a sufficient height before the noose tightens around the neck [21]. In rare instances, the head is not completely severed from the trunk so that the wound produced by the hanging device might be mistaken for a cut due to sharp force [12].

In victims of manual strangulation, an interposed necklace under the offender's hand may cause a linear skin abrasion later appearing as a dried streak similar to an incomplete ligature mark [12]. If a bead necklace is jammed by an overlying strangling device for a prolonged period of time, the bead pattern is seen within the pressure mark [12].

Haematomas from love-bites (sucking and/or biting kisses) often resemble fingertip bruises in manually strangled victims (Figure 39.7). However, the clinical symptoms characteristic of preceding pressure on the neck, including dysphagia, pain on moving the neck and dysphonia, are absent in such cases.

In victims of electrocution or lightning, patterned contact burns from necklaces may be vaguely similar to ligature marks. This kind of electrothermal skin lesion has been described most often in the context of lightning, where it is typically accompanied by metallization [12], but it occurs also in high-voltage electrocutions with arcing [1]. Metal objects worn on the body are heated and even made molten by the current, leaving a congruent burn on the underlying skin. Exceptionally, such electrothermal effects are seen after long-time exposure to current in the low-voltage range [17] (Figure 39.8).

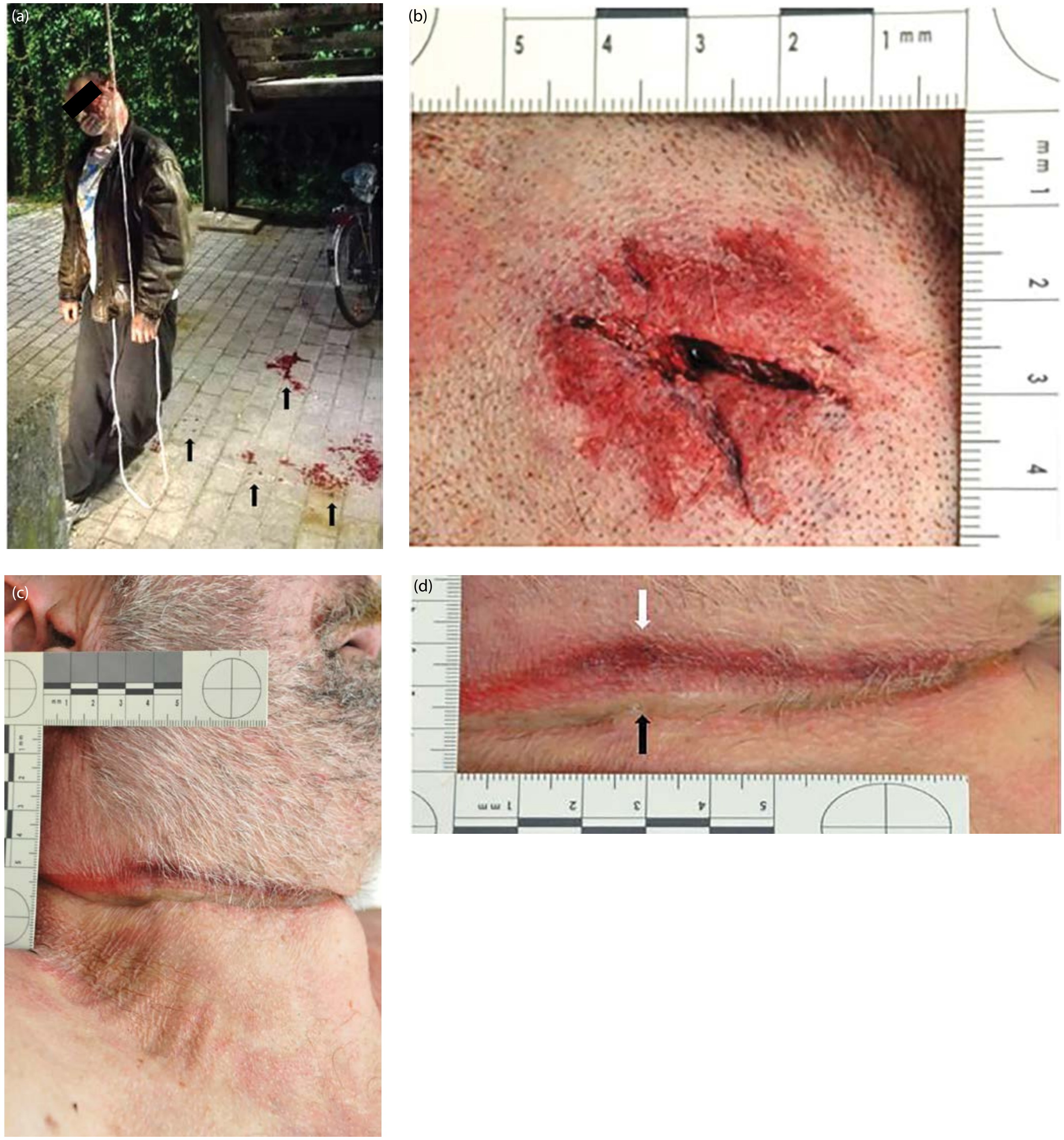

**Figure 39.6** Suicide by hanging. (a) The suspended body was found under a wooden bridge at nighttime. The disseminated blood stains on the pavement (arrows) raised suspicion of crime. The bleeding originated from a y-shaped laceration in the left parietal region above the hat brim line (b). (c), (d) Close inspection of the neck revealed two hanging marks although a single-turn noose had been used. (d) The upper mark was not deepened and was parched reddish-brown (↓), whereas the lower one constituted a furrow without discolouration as it was protected from drying by the overlying rope (↑). As a result of the medicolegal and criminal investigations, it had to be assumed that the first hanging attempt failed and entailed a fall to the ground that caused the laceration of the scalp.

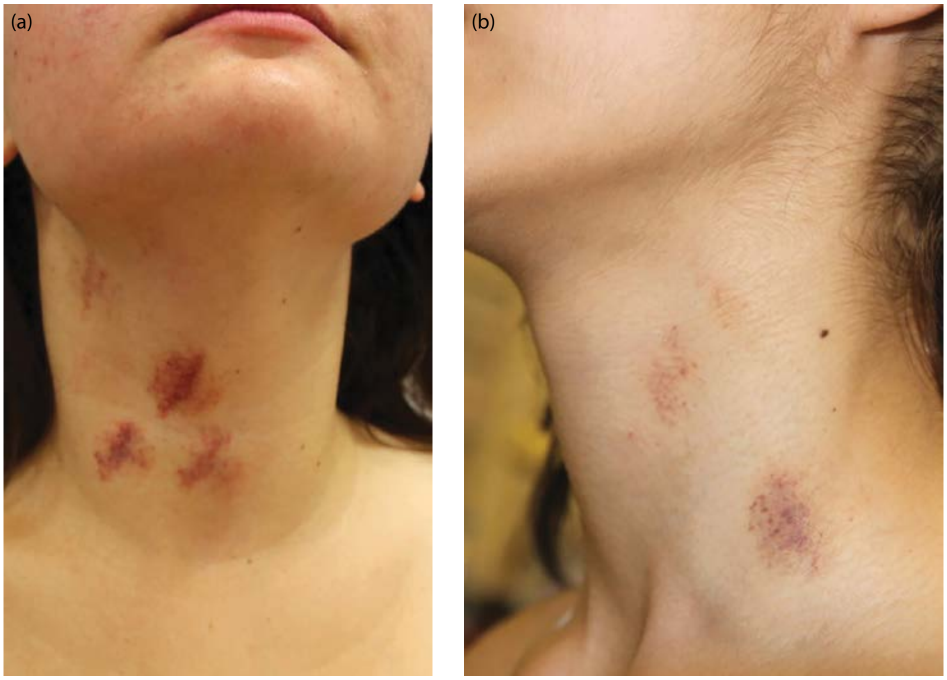

**Figure 39.7** (a), (b) Two young women showing so-called 'love bites' in the anterior and lateral neck regions imitating grip marks from manual strangulation.

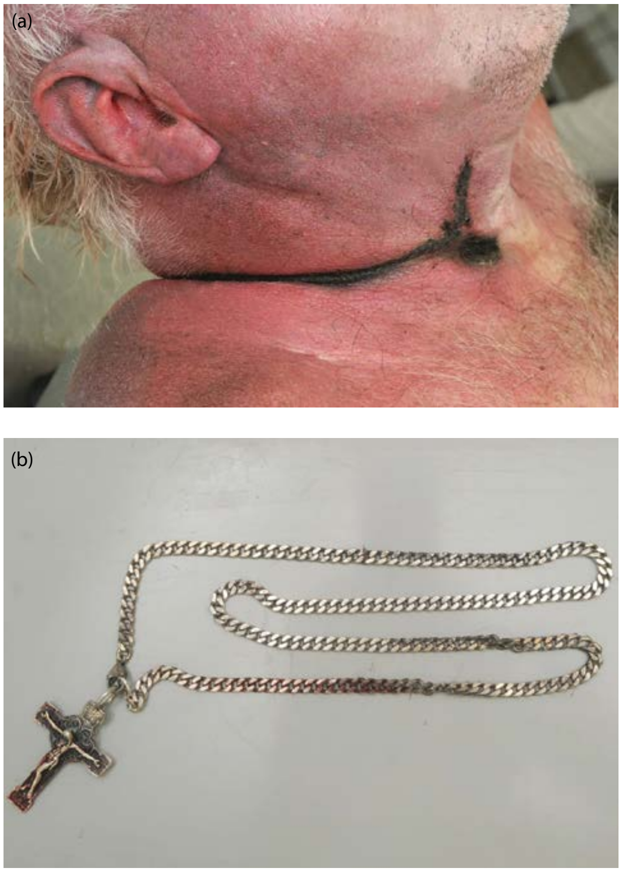

**Figure 39.8** (a), (b) Patterned contact burn along a silver necklace worn by a man who died from electrocution (long-term current flow, low-voltage range, accidental contact with a blank wire).

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